

Introduction of CNS

"From lectures of dr. Saif darif"

VISIT...

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Neurology

Organic

Psychologic

Functional classification of N. system:-

1. Sensory.
2. Motor.
3. Coordination
 - Cerebellum → movement
 - Basal ganglia → Rest

(coordination b/w ms. tone during rest).
4. Autonomic.
5. Mentality and Behavior.

Note:- We can't examine the nervous system in mental retard pt or who affected psychologically, so the 1st step should do before CNS examination is mentality test.

Anatomical classification of N. system:-

CNS

PNS

CNS Formed of 2 main parts:-

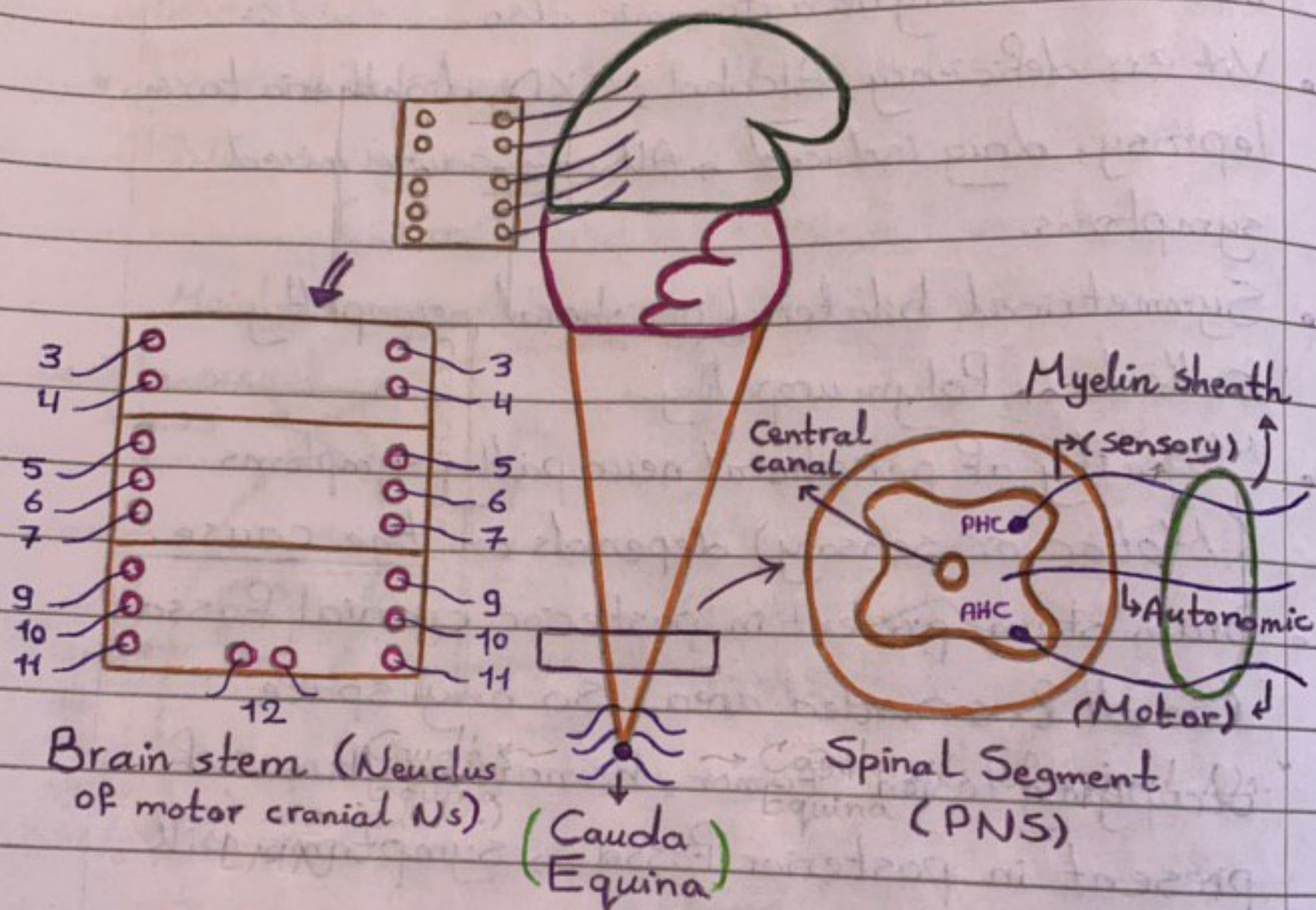
- Brain
- 2 cerebral hemisphere
 - Brain stem
 - Cerebellum
 - Basal Ganglia

- Spinal part
- Spinal cord
 - "31 segments"
 - Cauda Equina

Note:- During intrauterine life, the rate of growth of spinal cord is very slow while rate of growth of vertebral bones are very rapid.

- Vertebral growth continues till coccygeal & spinal cord ended at L₁ vertebra, form "conus medullaris".
- Below L₁ → lumbosacral plexus which's extension of spinal cord & called "Cauda Equina" → supplying the L.L, bladder & Rectum.
- Trauma, Disk prolapse...etc if affected below L₁ → will results to Cauda equina syndrome.
- "Peripheral nerves damage not spinal cord damage".
- Myelin sheath that covers CNS derived from Oligodendrocyte, while Myelin sheath that covers PNS derived from schwan cells (Histological classification)

Vit B₁₂ deficiency can affect both CNS & PNS.



- Every spinal segment have 2 pairs of nerves (Rt & Lt) → Totally 31 pairs.
- Every spinal nerve consists of : 1) Motor Fiber, 2) Sensory, 3) Autonomic (could be absent).
- Spinal neuropathy "Peripheral neuropathy" have mixed symptoms of spinal nerve lesion "Motor + Sensory & may be also autonomic".

- ex of mixed symptoms 2^{ry} to peripheral spinal nerve damage is diabetes → affecting sensory, motor and maybe autonomic also.
- Vit B₁₂ deficiency, Alcohol, CKD, diphtheria toxin, leprosy, drug induced → All can cause mixed symptoms.
- Symmetrical bilateral peripheral neuropathy called → Polyneuropathy.
- Majority of peripheral neuropathy symptoms (Motor or sensory) depends on the cause.
- Brain stem present in posterior cranial Fossa (small & crowded area) So any space occupying lesion "tumor, hematoma, Pus..." if present in posterior Fossa → symptoms will appear early.*
- For any posterior Fossa mass → Do MRI rather than CT. "more accurate".
- Urgent neuro imaging study is CT even in Posterior Fossa.
- Cranial nerves: 1, 2, 8 → Purely sensory.
10, 9, 7, 5 → Mixed.
3, 4, 6, 11, 12 → Motor.

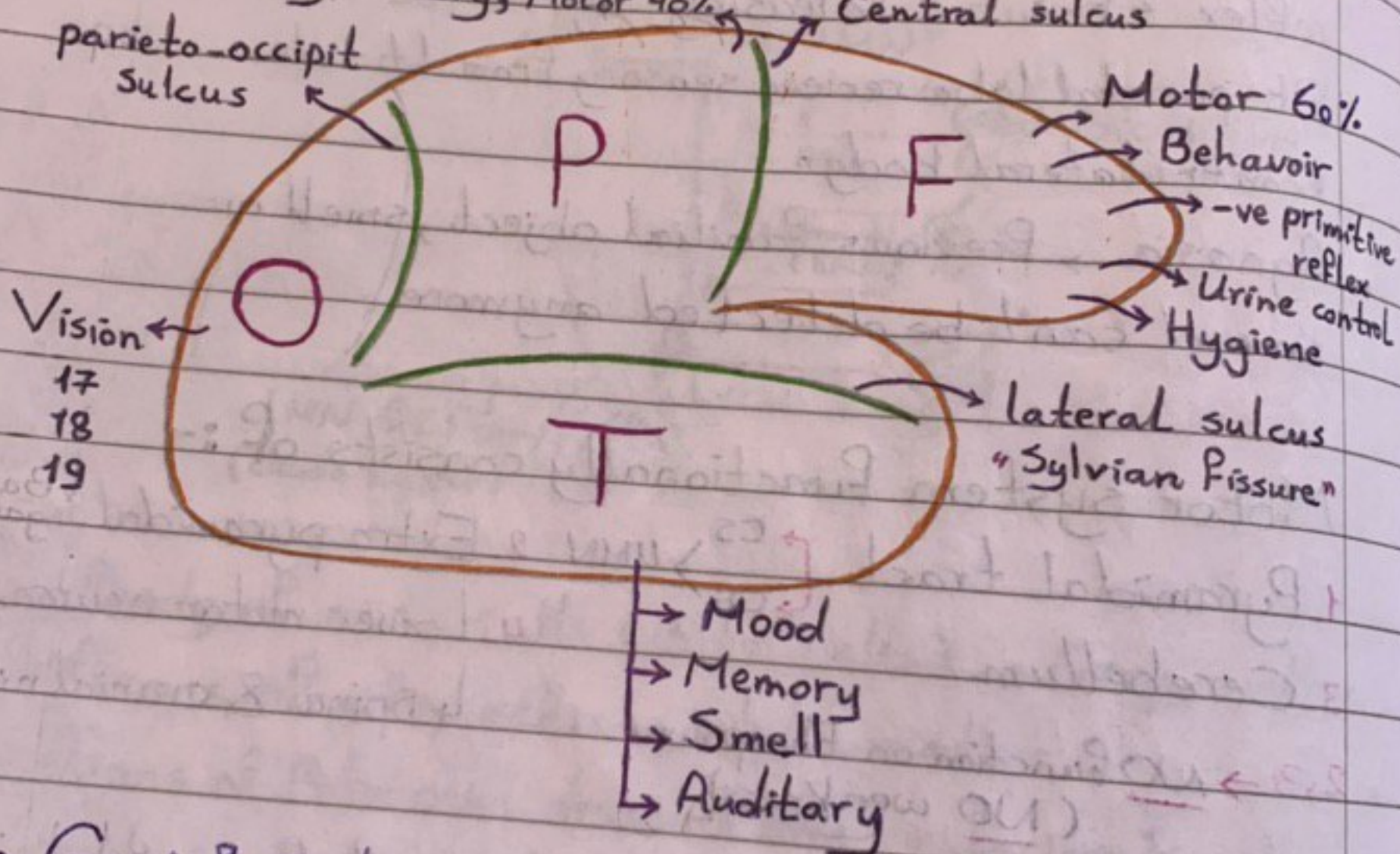
Note: Anosmia not consider as UMN or LMN lesion because olfactory nerve is purely sensory.

- In brain stem there's 9 motor nucleus.

PNS → any nerve outside the bone.

(cranial nerves & spinal nerves)

Mainly sensory, Motor 40%



- Gyri & Sulci present for ↑ surface area.

The main function of Frontal lobe is give motor

fiber order for contralateral side "upper & lower".

- Frontoparietal lobes give → Full ms. power.

So, if Frontal lobe is affected → paresis occur

not paralysis, because still have 40% from parietal lobe.

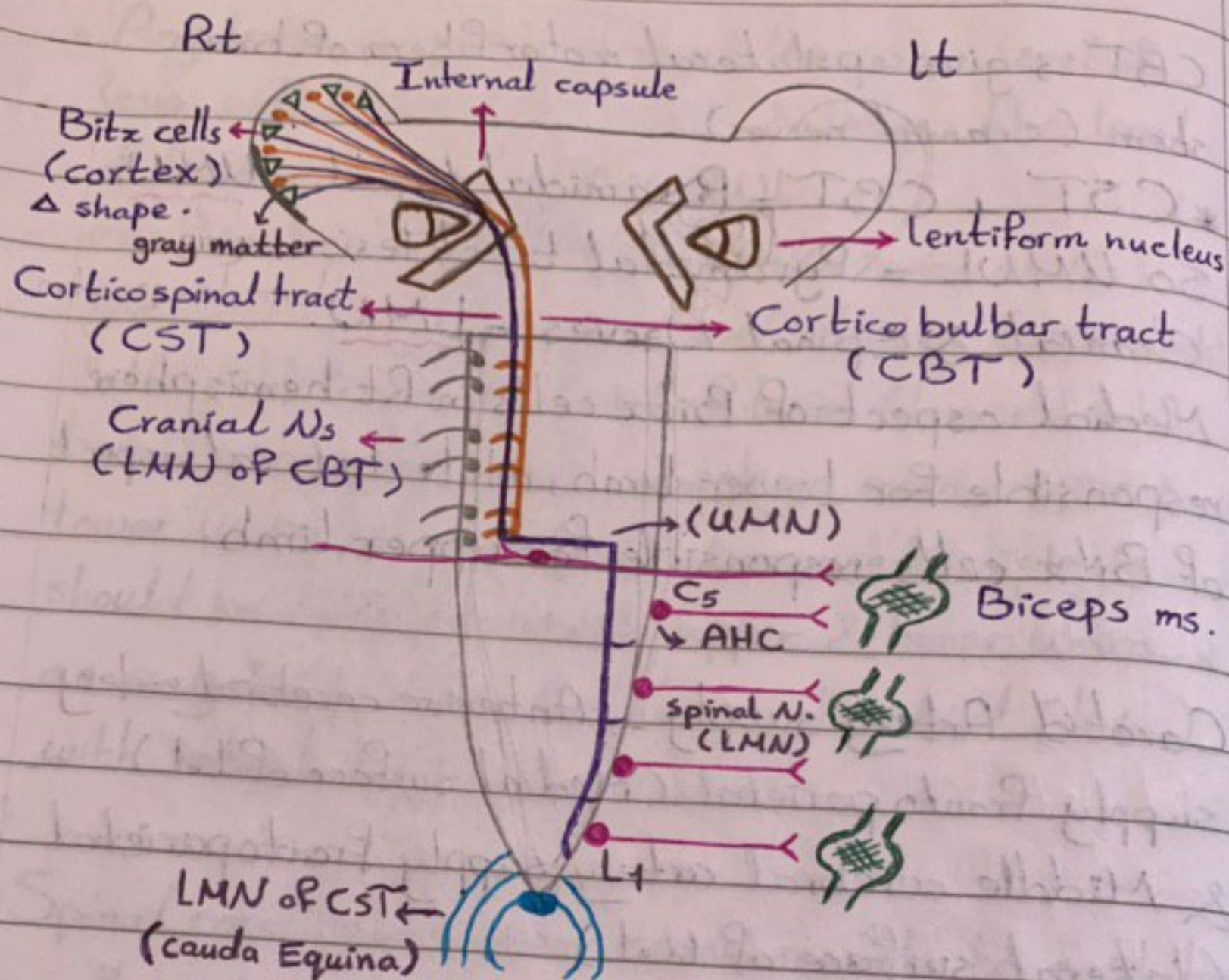
- Occipito-temporal infarction occurs due to posterior circulation occlusion, which's very rare. 5% affecting vision & memory → NO paralysis.
- Frontal lobe lesion → reappearance of primitive reflex "Frontal releasing sign".
Rt parietal lobe receive sensory from Lt side.
"contra lateral body".
- Agnosia → Previous familiar object, smell or sound can't be detected anymore.

Motor system functionally consists of :-

1. Pyramidal tract $\left\{ \begin{array}{l} CS \\ CB \end{array} \right\} \rightarrow UMN$
 2. Extra pyramidal.
 3. Cerebellum.
 4. Lower motor neuron.
↳ Spinal & cranial nerves
- 2, 3 ⇒ NO function on the power.
(NO weakness)

Examination of motor system called → تبارك
consists of :-

1. Tone
2. power
3. Reflex
4. Coordination



- Body of nerve cells (Bitz cells) $\rightarrow$ Grey matter.
Axons of nerve cells $\rightarrow$ White matter.
- Axons of Bitz cells descend over anterior $\frac{1}{3}$ of posterior limb of internal capsule $\rightarrow$ then continue to reach lower $\frac{1}{3}$ of medulla oblongata $\rightarrow$ then decussate to the contralateral side.
- For voluntary ms. contraction need 2 Nerves UMN & LMN.

- CBT → give ipsilateral motor fibers of brain stem (cranial nerves).

* CST + CBT = Pyramidal tract = UMN.

So UMN = Pyramidal tract lesion.

Cranial & Spinal Nerves = LMN.

Medial aspect of Betz cells in Rt hemisphere responsible for lower limb, while lateral aspect of Betz cells responsible for upper limb.

- Carotid Artery supply → Anterior cerebral artery supply fronto parietal (Medial surface of L.L).

& Middle cerebral artery supply frontoparietal (lateral surface of U.L).

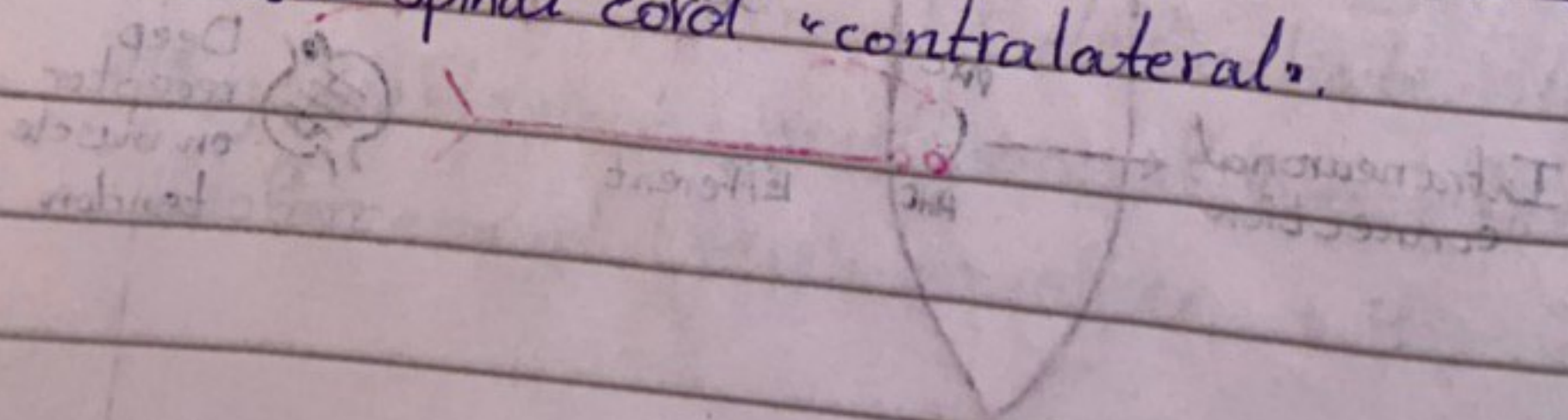
Posterior cerebral supply occipito temporal.

- Capsular lesion lead to hemiplegia (upper & lower) starting at the same time.

Cortical lesion lead to hemiplegia (one limb starting before other). because necrosis associated with edema → lead to indirect compression on other site.

- Any lesion in spinal cord at the same level of lesion will results to LMNL & below the level of lesion will results to UMNL.
ex: Brachial Plexus lesion (AHC lesion at C5) give LMNL at the same level of lesion (upper limb) & UMNL below the level of lesion (lower limb). but it's not consider as hemiplegia even upper & lower limbs are paralysed, because hemiplegia should be UMNL in both upper & lower limbs as any lesion above C5 (above brachial plexus) with intact LMN.

- Spinal cord lesion (after cross) results to ipsilateral paralysis & hemiplegia. while any lesion before cross as cortical lesion results to contralateral hemiplegia.
- Hemiplegia with normal cranial nerves → lesion at the spinal cord "Below brain stem".
Hemiplegia with affected cranial nerves → lesion above spinal cord "contralateral".



Function of UMN & LMN :-

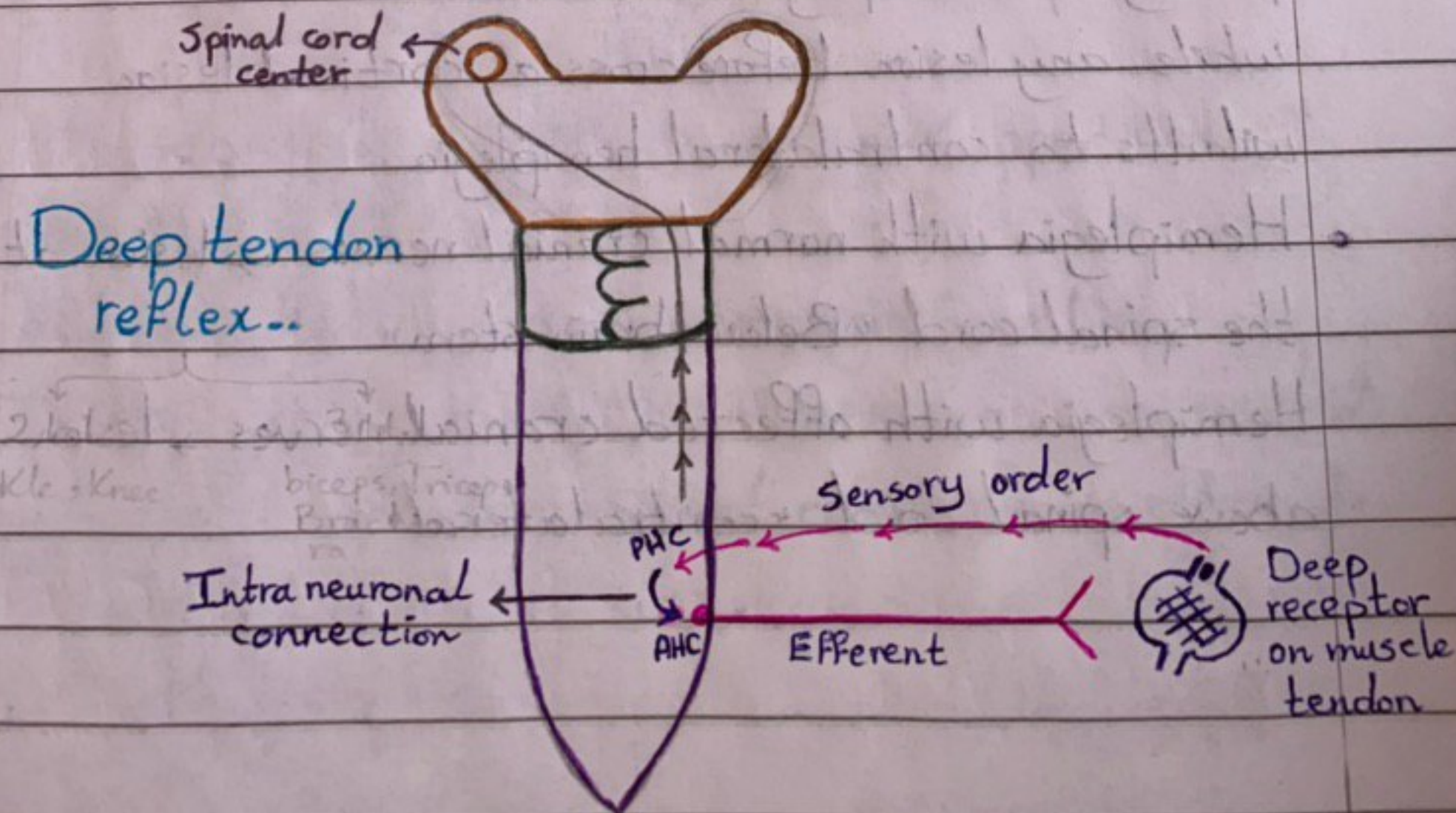
UMN (CST)

1. Transmission of action potential from cortex to AHC.
2. Inhibit of ms tone & Reflex.

LMN (AHC & Peripheral nerves)

1. Transmission of action potential from AHC to neuromuscular junction.
2. Generate of ms tone & Reflex.

Note: AHC have automaticity * generate small action potential → lead to continuous weak involuntary contraction during rest called → Ms tone.



Deep tendon reflex

- When we do deep tendon reflex by hammer
 → sensory order transmit from deep ms. receptor to the PHC of the same segment level & to the spinal cord center (send afferent message → there's ms stretching) to protect the muscle. The impulse then transmit from PHC to AHC directly by intra neuronal connection (no impulse transmission to the cortex). → lead to involuntary ms contraction (Deep tendon reflex)

Root values of deep tendon reflex

↓
2 L.L

- Ankle S₁, S₂
- Knee L₃, L₄

↓
3 U.L

- Biceps C₅, C₆
- Brachioradialis C₅, C₆
- Triceps C₇, C₈

- UMN → Weakness, Hypertonia, Hyperreflexia
- LMN → Weakness, Hypotonia, Hyporeflexia

Note: Reinforcement differentiate b/w hyporeflexia & areflexia. if even after reinforcement there's no reflex → it's areflexia

Finding	UMNL	LMNL
1. Weakness	Pyramidal distribution "affecting more muscles".	Segmental distribution.
2. Tone	Hypertonia (spasticity)	Hypotonia (Flaccidity)
3. Deep Reflexes	Hyperreflexia (Brisk reflex)	Hyporeflexia or Areflexia.
4. Superficial Reflexes	Disappear except plantar.	Normal or disappear.
5. Plantar Reflexes	+ve Babinski Sign.	Equivocal response.
6. Ms. Wasting	+ late & mild	++ early & severe
7. Fasciculation	Absent	Present (Late)
8. Pathological Reflexes	++	-

Notes :- Ms wasting present in both UMNL & LMNL, but in UMNL it's milder & late because UMNL have hypertonia in form of spasticity which's preserve ms. bulk $\rightarrow$ $\downarrow$ wasting.

D/D of Equivocal superficial reflex :-

1. Diabetic neuropathy (stock glove sensory loss).
2. Thick skin.

Fasciculation

- Group of ms twitching due to chronic deinnervation with partial reinnervation caused by LMNL or physiological without ms. weakness.

- Ms. twitching after stimulation
- Can be seen by naked eye.

Fibrillation

- It's deinnervation of ms - acute or chronic lead to pathological stress & Disturbance of Ca^{+2} storage caused by UMNL & LMNL.

- NO sign, detected by EMG.
- Can't be seen by naked eye.

- Notes :-**
- IF you find areflexia over ankle tendon even after reinforcement but there's babinski sign +ve in the same leg $\Rightarrow$ it's a mixed lesion.
 - IF you find Fasciculation over deltoid ms & Exaggerated reflex over biceps & Brachioradialis $\Rightarrow$ it's mixed lesion in upper limb.
- (Absence of deep reflex + Babinski sign +ve = Mixed lesion)

- After examination of L.L & you find weakness in both L.L → your doctor ask you where's the site of lesion?

Told him i don't know, i have to examine the upper limb first:

if the upper limb is normal → lesion is below T₁.

if the upper limb is involved → lesion is above brachial plexus.

- Hypertonia due to pyramidal tract lesion called Clasp Knife spasticity.

Hypertonia due to extra pyramidal tract lesion (Basal ganglia) is rigidity in type (resistant all over the range of joint movement).

- Clonus: It's indicate severe UMN L.

Must be there's 3 or more plantar Flexion to say there's +ve clonus.

D/D → 1. Severe UMN L

2. Preeclampsia

3. Hormonal disturbance

4. Renal impairment

5. Uncontrolled HTN

- Ankle clonus doesn't confirm UMNL & if -ve doesn't exclude.
+ve Babinski sign confirm UMNL, but if it's -ve doesn't exclude.
- * In general we can't exclude any disease by absence of any clinical sign in medicine.
- UMNL less severe than LMNL, because paralysis of LMNL is irreversible due to no ms. tone + weakness → Can't stand against gravity. while UMNL it have weakness also but hypertonia help pt to stand.
Also paralyzed hypotonic pt is risky of Bedridden complications as bed sore, DVT, Pulm. embolism (↑ mortality rate in early stage).

Note :- CVA pt is risky of aspiration pneumonia. So once pt comes, examine gag reflex by tongue depressor → if he lose it (-ve gag reflex) don't give pt anything orally to avoid the risk of aspiration pneumonia.

D/D of UMN:-

1. Trauma (head trauma, back trauma, Disk trauma)
There's a Hx of trauma with *acute to sudden onset.

2. Tumor "1st → rare" or "2nd → more common".

metastasis from other sites to → white matter.
& symptoms usually gradual in onset + Features of ↑ ICP + Fever & wt loss.

3. Infection → Meningitis (Bacteria + T.B),

Encephalitis (viral), Brain abscess

All of them can have fever, neck stiffness, Blurred vision & photophobia (signs of meningeal irritation but encephalitis can also lead to confusion.

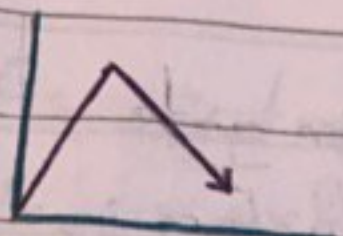
Brain abscess associated with strong weakness because of space occupying lesion compression (collection of pus).

4. Inflammation → Demyelinated ex: M.S

Notes :- The function of myelin sheath is protect the nerve & help in conductivity & velocity of action potential.

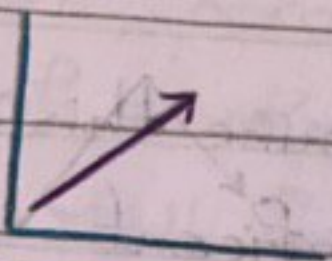
- Demyelination → ↓ velocity of conduction but the power of conductivity is normal.
- If the axon is involved → velocity & power of conductivity will ↓.

- In all demyelinated diseases, usually the axon is preserved & healed with fibrosis.



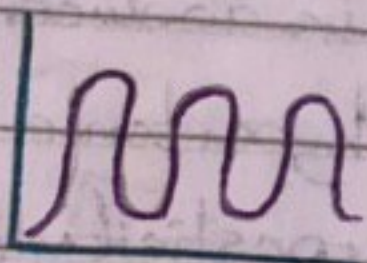
(Traumatic course)

Symptoms start huge then subsided → *regressive course



(Tumor course)

* Progressive course



(M.S course)

Chronic course with relapse & remission

Note :- Infection have short duration (No course).

5. Vascular → CVA "accidental cerebral event 2^{ry} to vascular disease either occlusion or Hge.

CVA symptoms should be * sudden in onset.

↳ ischemic → painless without headache.

↳ Hemorrhagic → painful with headache.

Differentiate b/w them by CT-scan

UMNL :- 1. Weakness in Proximal > Distal.

2. Flexors > Extensors.

3. Abductors > Adductors.

ex: CVA have circumduction gait.

Note :- Abductors weaker than adductors in other diseases as proximal myopathy, Dermatomyositis, acromegally etc.

- **Example of presentation :-** While examine this pt gentle man / lady, both L.L had significant weakness. distribution of weakness in Proximal more than distal, Flexors > Extensors, in abduction more than adduction, associated with hypertonia all over the joints bilaterally in form of spasticity & exaggerated of deep tendon reflex "Hyperreflexia" & Extensor plantar reflex bilateral "Babinski sign".

The coordination can't be performed because of weakness.

This Finding is going with UMNL For D/D

LMNL:- 1. Distal > Proximal

2. Extensors > Flexors

3. Abduction > Adduction

Associated with hypotonia in form of Flaccidity & hyporeflexia or areflexia according to the finding & Plantar reflex normal or equivocal. The coordination can't be performed because of weakness. This findings are going with LMNL For D/D.

D/D of LMNL:-

1. Trauma (Spinal cord trauma, Bone Fracture.)

2. Tumor → 1ry or 2ry.

3. Infection → Myelitis, Polio (AHC), T.B (Pot's disease)

4. Inflammation → Demyelinated (GBS) → LMNL

acute in onset with no relapse & remission course

5. Vascular → Anterior spinal artery thrombosis. common & acute. (↑ with hypercoagulopathy state).

D/D of Mixed:-

1. Friedreich's ataxia → AR, chromosome 9 pt young in age, ♂ = ♀, has D.M & H.F or DCM die after 20-30 yrs. "most commonly due to H.F."

& associated with high arched Foot.

- All parts of CNS & PNS are involved in Friedreich's ataxia.

2. Vit B₁₂ deficiency → Sub acute combined degeneration. (Anemia + Neurological manifestation)

↓ vit B₁₂ → Mudness → hypothyroid like symptom.

- It's called SACD because it's affect motor & sensory & mainly start by sensory.

vit B₁₂ deficiency affect :-

1. Pyramidal system.

2. Posterior column.

3. Peripheral Nerve.

- Vit B₁₂ & B₉ needed For DNA replication when ↓ in Blood → cytoplasm divided but nucleus don't → Megaloblastic anemia.

MCC of vit B₁₂ is Autoimmune gastritis

(Pernicious anemia).

Vit B₁₂ deficiency > 90 days → irreversible

Vit B₁₂ deficiency < 90 " → reversible

So Rx early as possible.

3. Motor neuron disease (MND) "Atrophic lateral sclerosis" → affecting motor system only.

4. Tabes dorsalis.

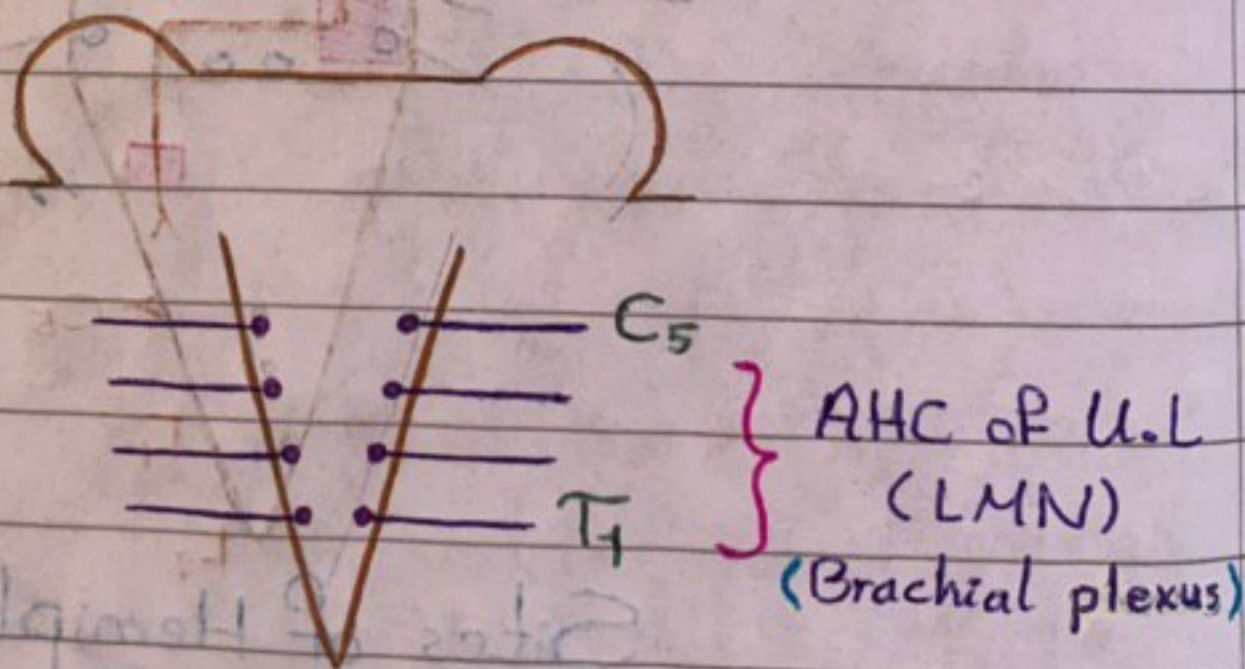
5. Syringomyelia $\rightarrow$ affecting AHC & CST laterally.

6. DM amyotrophy.

Note:- When you find mixed lesion ex: Absent Ankle reflex + plantar reflex +ve $\rightarrow$ told your doctor i have to examine sensory system $\rightarrow$ if it's normal $\rightarrow$ pt have MND.

Localization of Hemiplegia...

Def:- Paralysis of one side of the body (Rt or Lt) by UMN.

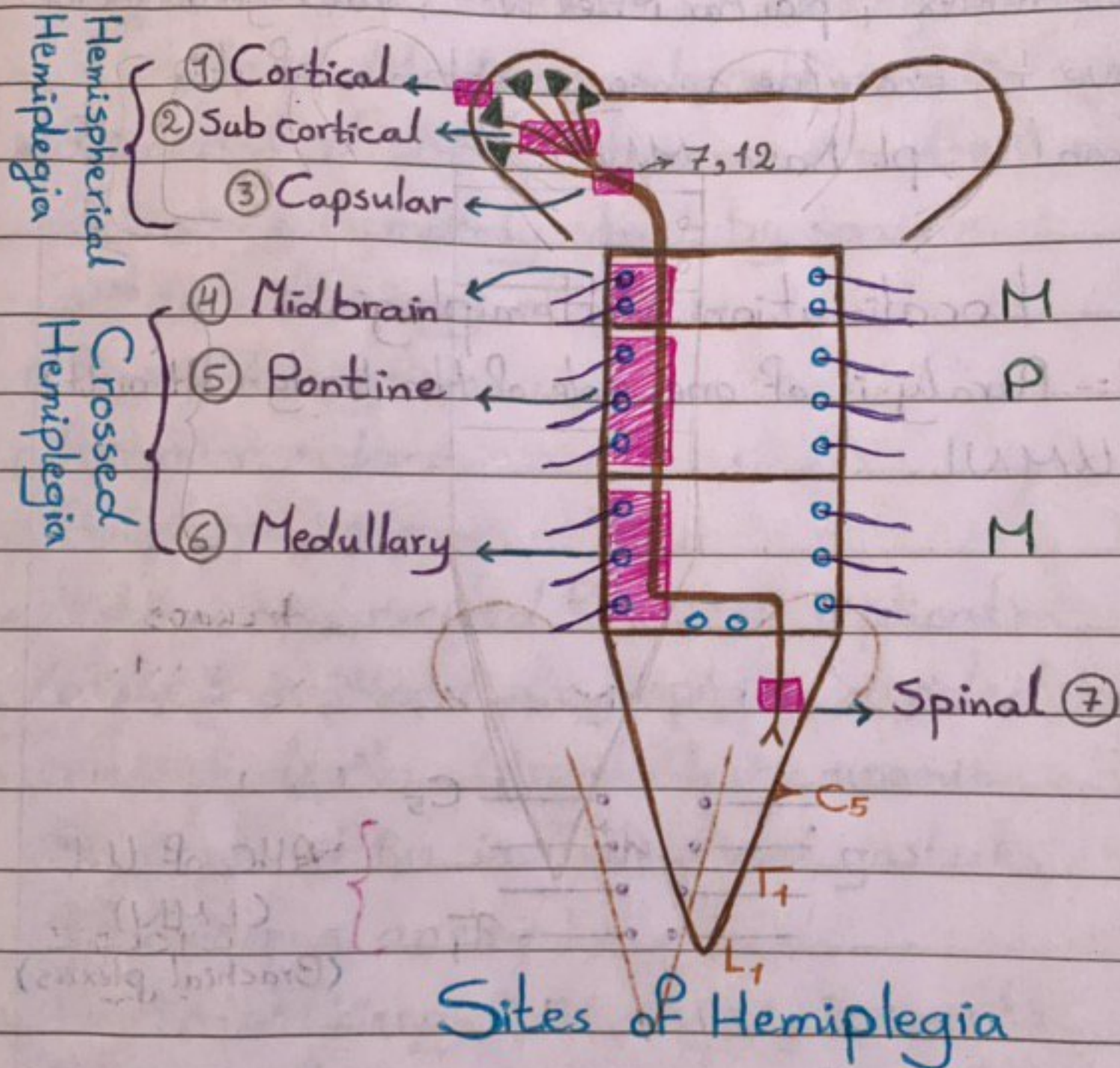


• To do UMN for the U.L $\rightarrow$ the lesion should be above C5.

Lesion below C5 $\rightarrow$ LMN in U.L with UMN in L.L $\rightarrow$ it's not hemiplegia even both limbs are paralyzed.

Causes of hemiplegia :- (As D/D of UMN). 13

1. Vascular (MCC $\rightarrow$ ischemic 85%)
2. Tumor
3. Trauma
4. Infection
5. Inflammation

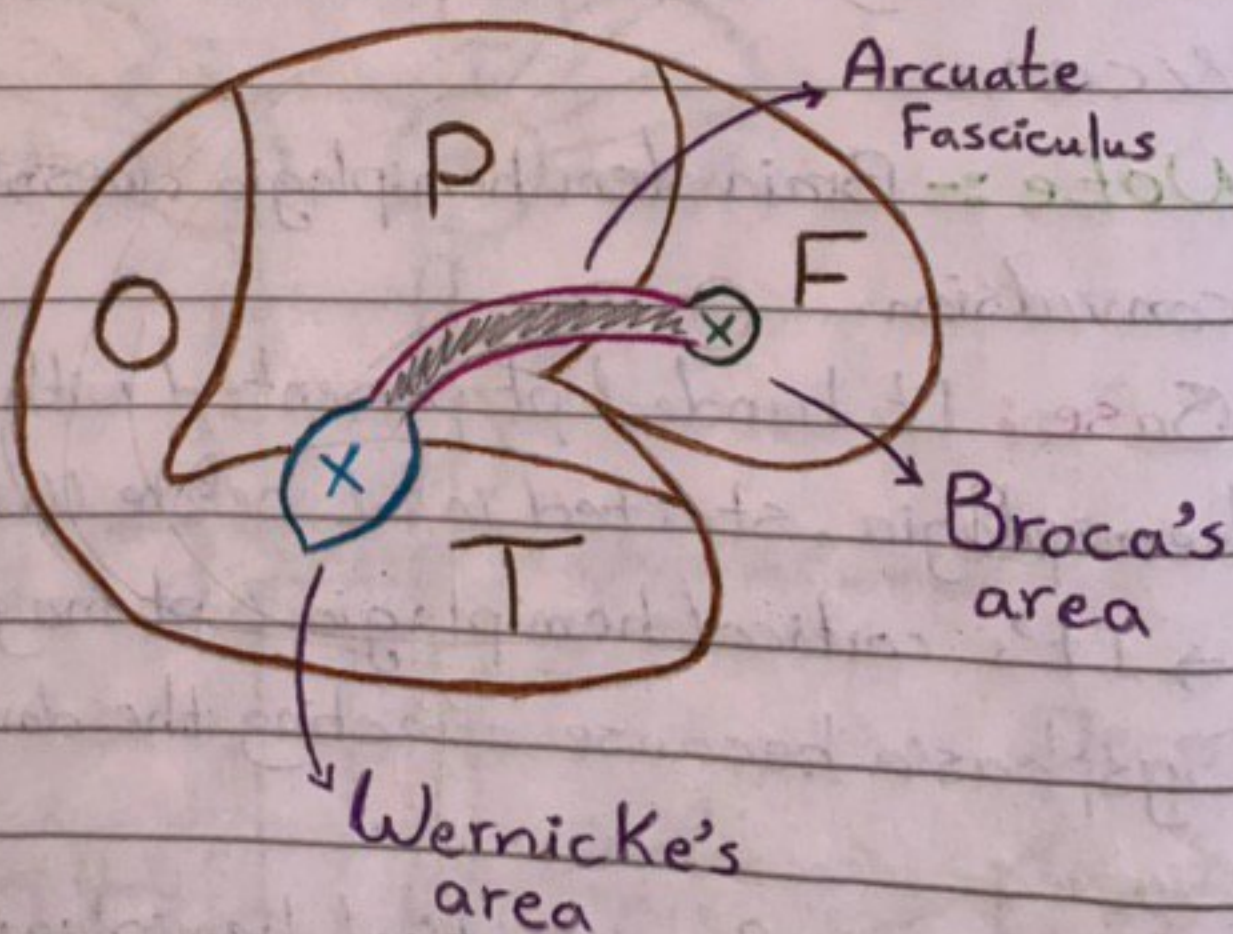


Sites of Hemiplegia

Note :- Hemiplegia termed according to paralysed limb, not the site of lesion.

ex: Lt side hemiplegia means the lesion could be ipsilateral or contralateral.

- Lesions From 1 $\rightarrow$ 6 lead to contralateral hemiplegia.
- 7 $\rightarrow$ lead to ipsilateral hemiplegia "by hemi cut above C5".
- Brain stem hemiplegia $\rightarrow$ Poor prognosis especially medullary lesion due to loss of gag reflex.
- Crossed hemiplegia means there's contralateral side hemiplegia + ipsilateral Face involved. occurs only with brain stem.
- All cranial nerves in spinal hemiplegia are normal $\rightarrow$ & confirmed by sensory examination.



Dysphasia / Aphasia : Definition :-

It's Inability to speak or understand mother language. (affecting dominant hemisphere)

- Wernicke's area lesion → inability to understand native mother language. because it's Function is sensory "comprehension".
- Broca's area lesion → inability to speak because it's Function is motor "Fluency".

- Cortical hemiplegia
- Cortical hemiplegia starting in one limb before other. ex: medial cortical lesion lead to necrosis in L.L then by edema & compression affecting U.L. it's usually associated with convulsion, confusion & coma.

Note:- Brain stem hemiplegia doesn't lead to convulsion.

Case: Lt handed pt presented with Lt side hemiplegia, started in L.L before U.L...

⇒ It's cortical hemiplegia & pt may have Dysphasia because affecting the dominant side.

- Presentation of subcortical hemiplegia is similar to cortical.

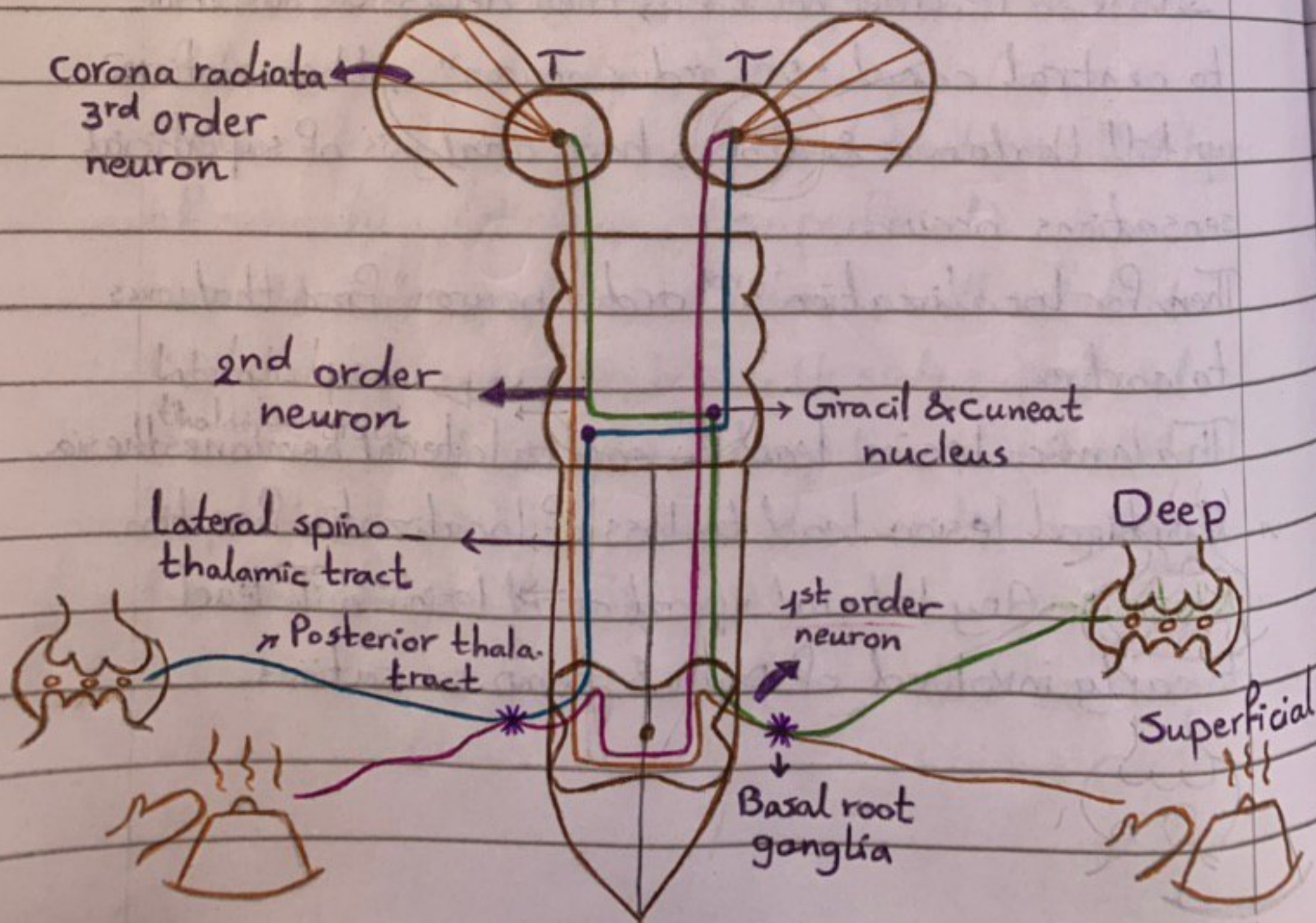
- Capsular hemiplagia have sudden onset of weakness in both limbs at the same time without convulsion.

7, 12 cranial nerves can be involved at the same side of lesion.

- The Most common site of hemiplagia is
* Capsular & the most common artery occluded is MCA.

The MCC of capsular is ischemic occlusion.

Sensory system



* Superficial

1. Pain

2. Temp

3. Touch

Crude Fine

* Deep

1. Vibration

2. Tension

3. Pressure

4. Position

- Fine touch is most rapid sensation (more than pain & temp) because it's well myelinated.

- Superficial peripheral N. course: ascending as lateral spinothalamic tract → Dorsal root ganglia → PHC "1st order neuron" → then decussate anterior to central canal "2nd order neuron" → then continue up till thalamus & stop → here analysis of superficial sensations occur.

Then for localization "3rd order neuron" From thalamus to cortex.

Thalamic lesion lead to contralateral hemianesthesia.

Cortical lesion lead to loss of localization function.

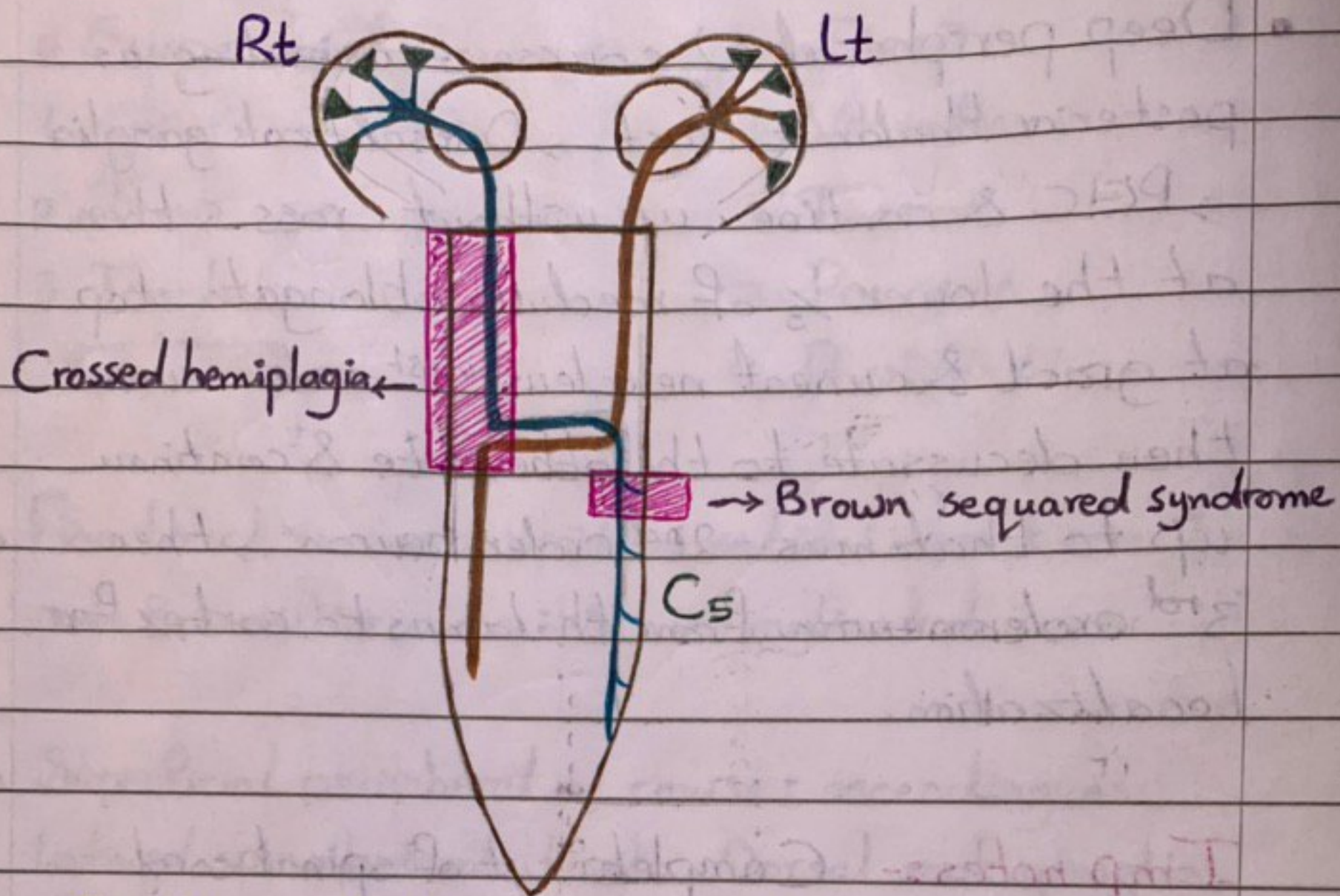
Note :- Any lateral spinal cord lesion will lead to early involved of pain & temp. sensations.

- Deep peripheral N. course :- ascending as posterior thalamic tract $\rightarrow$ Dorsal root ganglia $\rightarrow$ PHC & continue up without cross $\rightarrow$ then at the lower $\frac{1}{3}$ of medulla oblongata stop at gracil & cuneat nucleus "1st order neuron". then decussate to the other side & continue up to thalamus "2nd order neuron" $\rightarrow$ then "3rd order neuron" from thalamus to cortex for localization.

Imp notes:- Complete cut of spinal cord lead to loss of all sensations below the level of lesion.

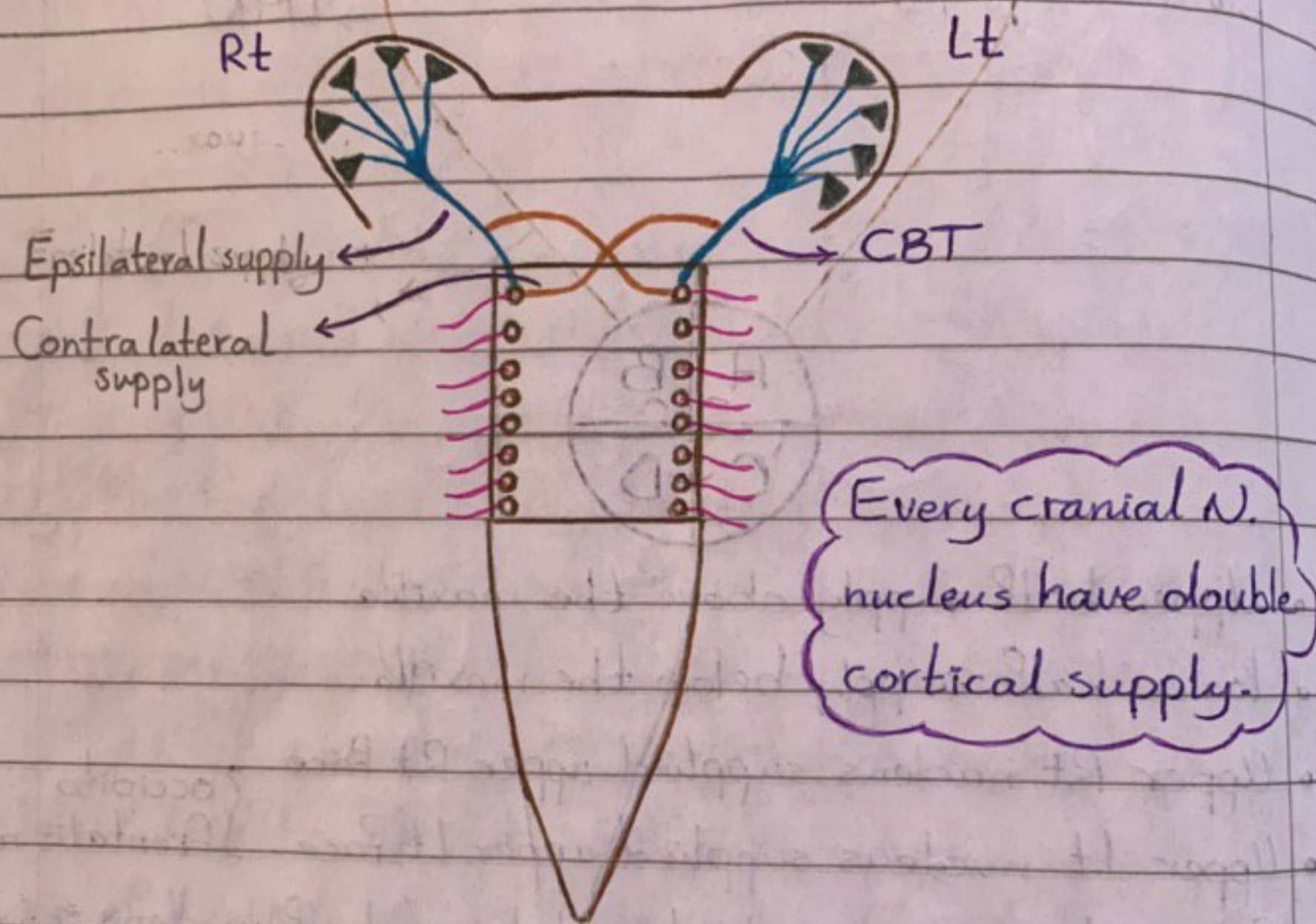
Hemicut of spinal cord lead to loss of 2 types of sensation (1 deep, 1 superficial).

ex:- Rt side spinal cord lesion $\rightarrow$ will results to Rt side deep sensation loss + Lt side superficial sensation loss (ipsilateral deep loss + contralateral superficial loss) & called dissociation sensory loss if combined with motor.

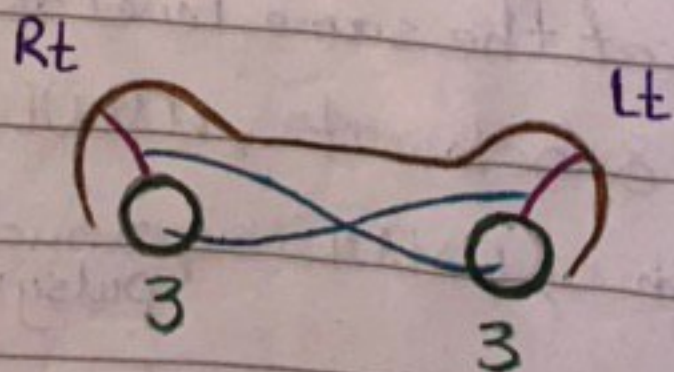


- Any hemicut above C5 in spinal cord will results to ipsilateral weakness, ipsilateral deep sensation loss + contra lateral superficial loss.
(2 sensory + 1 Motor) → * Brown sequared syndrome.
- Any brain stem lesion will results to contralateral hemiplegia + ipsilateral cranial Ns involvement + contra lateral hemianesthesia (superficial + deep).
- Capsular lesion lead to contralateral hemiplegia + contra lateral hemianesthesia + 7th & 12th CN involvement.

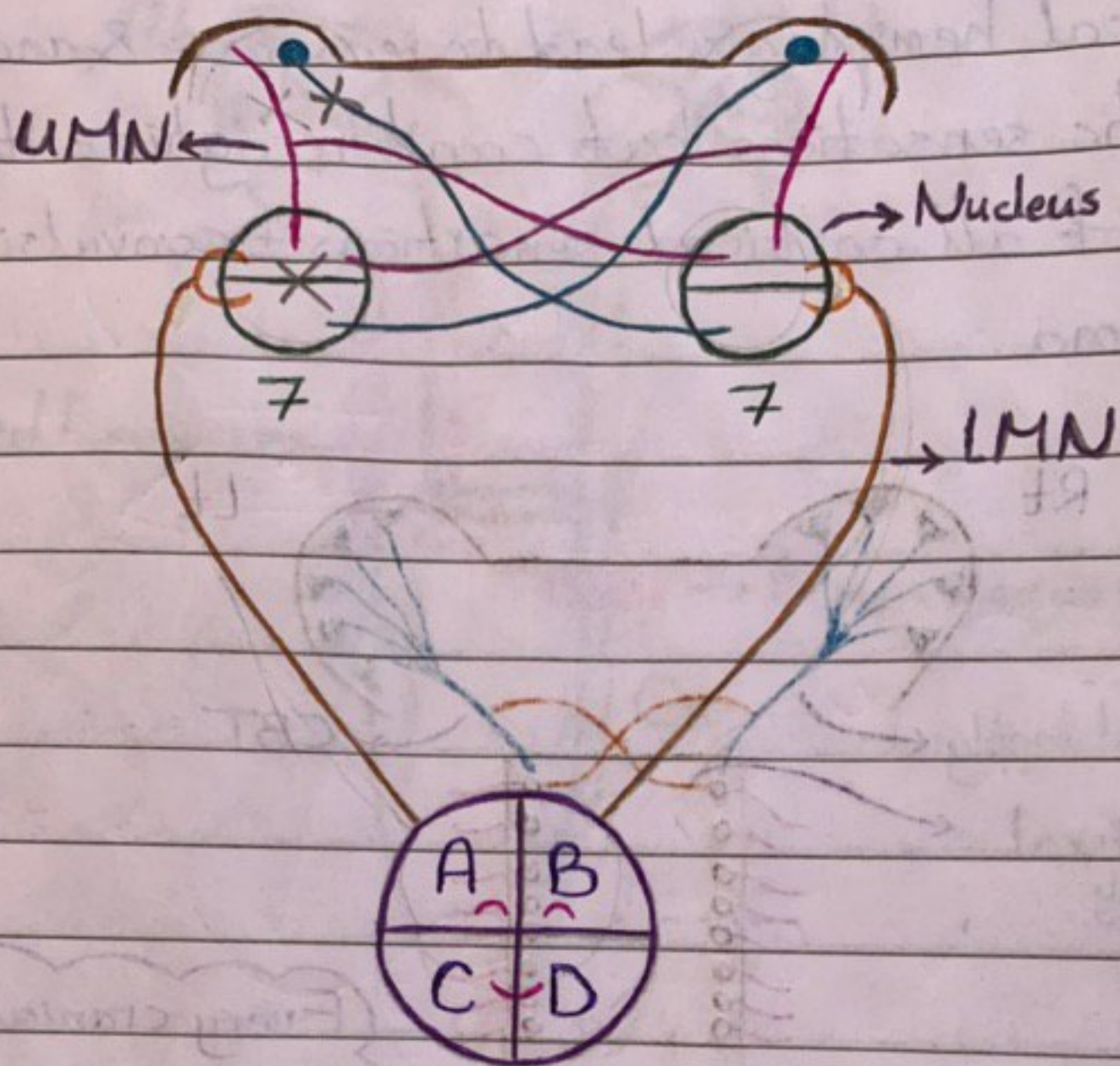
- Cortical hemiplegia lead to recognize & analyse somatic sensations but can't localize it + loss of all cortical sensations + convulsion or coma.



- Any cortical or capsular lesion in one side → Cranial Ns will not be affected except 3, 7, 12 CN because supplied unicortically.



ex: oculomotor Nerve have Epsilat & contralat. supply.



- Upper half supply above the maxilla.
- Lower half supply below the maxilla.
- Upper Rt nucleus supplied upper Rt Face.
- Upper Lt nucleus supplied upper Lt Face. } occipito Frontalis ms.
- Rt cortex give supply to Lt lower half nucleus. "contra lateral".
- Any lesion in nucleus or below → results to LMNL
- in the lower & upper parts of the face & sometimes called bulbar palsy. (ipsilateral hemifacial weakness)
- Any lesion in Brain stem → at the same level of lesion will results to LMNL. & below it → UMNL.
- Any lesion above Brain stem → UMNL "supranuclear palsy".

- Cortical lesion affect lower half of nucleus only because have unicortical supply.

So always UMNL of 7th N. palsy will results to contralateral weakness in lower half of Face.

{ LMNL of Facial N → always ipsilateral involve upper ms of the Face. & UMNL of Facial N is always contralateral. }

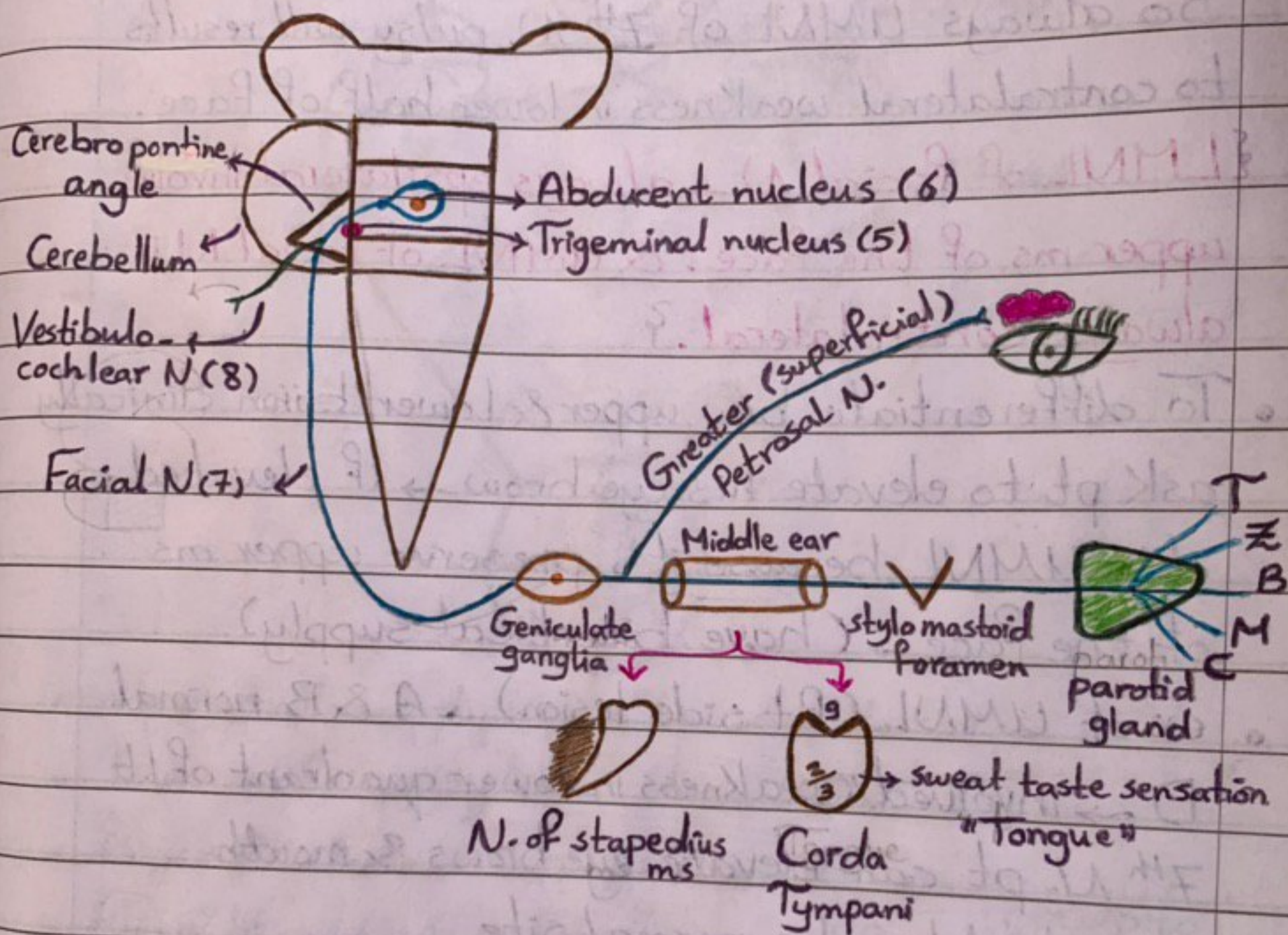
- To differentiate b/w upper & lower lesion clinically ask pt to elevate his eye brow → if elevated, it's UMNL because it's preserve upper ms of the Face → (have bicortical supply).

- ex of UMNL (Rt side lesion) → A & B normal D → involved "weakness in lower quadrant of Lt 7th N. pt can elevate eye brows & mouth deviated to the normal site.

- ex of LMNL (Rt side lesion) → Rt side Face paralysis (A & C) ipsilateral, pt can't elevate eye brow of affected side & mouth deviated to normal site.

Note :- Term "Palsy" used when there's muscle involvement.

Course of Facial N...



Q:- 1. Is this pt has Facial palsy?

2. Is UMNL or LMNL?

3. IF it's LMNL "which's more common than

4. UMNL" → where's the site of lesion?

5. What's the cause?

6. What's the Rx?

Answers :- 1. By mouth or lips deviation
if it's deviated to the Rt side $\rightarrow$ Lt Facial palsy.

2. Ask pt to elevate eyebrows "as mentioned before"

Contents of cerebro-pontine angle :-

1. Facial N.

2. Vestibulocochlear N.

3. Trigeminal nucleus.

Note :- When you examine the Facial nerve, examine 6 & 8 nerves also to detect the site of lesion. ex $\rightarrow$ if 6. N is involved $\rightarrow$ it's pontine cause

$\hookrightarrow$ if 8. N is involved $\rightarrow$ it's cerebro pontine angle cause $\rightarrow$ & most commonly due to acoustic neuroma $\rightarrow$ Benign tumor of shwan cells of 8. N.

- 7. N relay in geniculate ganglia $\rightarrow$ which's the site where HZV stay dormant until the immunity is $\downarrow$ $\rightarrow$ reactivated again.

HZV prefer to stay inside the ganglia because it's temperature is lower than body temperature.

- Corda tempani $\rightarrow$ give sensation anterior $\frac{2}{3}$ of tongue "sweet taste".

Glossopharyngeal N $\rightarrow$ give sensation for posterior $\frac{1}{3}$ of the tongue "Bitter taste".

- The function of N. of stapedius ms is to reduce perceived intensity of impulse sound. when it's damage results to hyperacusis* (unusual tolerance to ordinary environmental sounds).
- Stylomastoid Foramen is very narrow area, if pt exposed to cold air $\rightarrow$ will lead to edema formation & compression over 7.N $\rightarrow$ tenderness & 7.N pulsy. $\Rightarrow$ called Bell's pulsy "idiopathic, usually start within days & before it pt had common cold or exposed to cold air with feeling of tenderness behind the ear.
- The best line of management is reassurance.*
Relieve edema by $\rightarrow$ Steroid as early as possible.
Acyclovair For $\rightarrow$ Dormant HZV. + physiotherapy.
(90% of pts recovery within 2 wks).

The MCC of LMNL of 7.N is $\rightarrow$ Bell's pulsy.

Note :- Gum chewing is not a part of physiotherapy because it moves muscle of mastication which's not supply by Facial N.

Facial N divided inside the parotid gland into superficial & deep parts with 5 branches but it doesn't supply the parotid gland.

- The 5 terminal branches of 7.N inside the parotid are :- Temporal, Zygomatic, buccal, Mandibular, Cervical. (Motor branches).
- Causes of 6 & 7 CN lesion → Pontine Hge, Pontine tumor, Pontine compression.
- Causes of 5, 7 & 8 CN lesion → Acoustic neuroma, HZV infection, Otitis media, Fracture base of skull or Bell's palsy, Parotid tumor, Mumps → any parotid enlargement cause can lead to Facial palsy. also causes of peripheral neuropathy as DM.

Note:- Sarcoidosis affecting Parotid gland and lead to uveoparotid Fever (Heerfordt syndrome). & causing bilateral parotid enlargement → lead to Bilateral Facial palsy.

- During examination of Facial N → tell your doctor for detect the site of lesion, i have to examine :-
 1. Behind the ear → Bruises, tenderness, scar.
 2. Inside the ear → HZV vesicles, discharge of O.M, hearing → hyperacusis "lesion after the branch."
↳ Diffness "lesion before the branch".
 3. In front of the ear → Parotid, scar.
 4. Tongue examination → taste, & examine the palate for HZV vesicles.

4. Eye examination → Tear.

Rx of Facial N. palsy according to the cause.

Sensory System

1. Somatic sensations:

I. Superficial sensations: pain, temp, Touch (Deep)!

II. Deep sensations (Proprioceptive): Vibration, Tension, position, Pressure.

II. Cortical sensations:

Imp note: - The difference b/w animals & human beings sensations is cortical sensation which's present only in human beings.

Both analysis & localization of cortical sensations take place in cortex.

* Types of cortical sensations:-

1. 2 points discrimination.

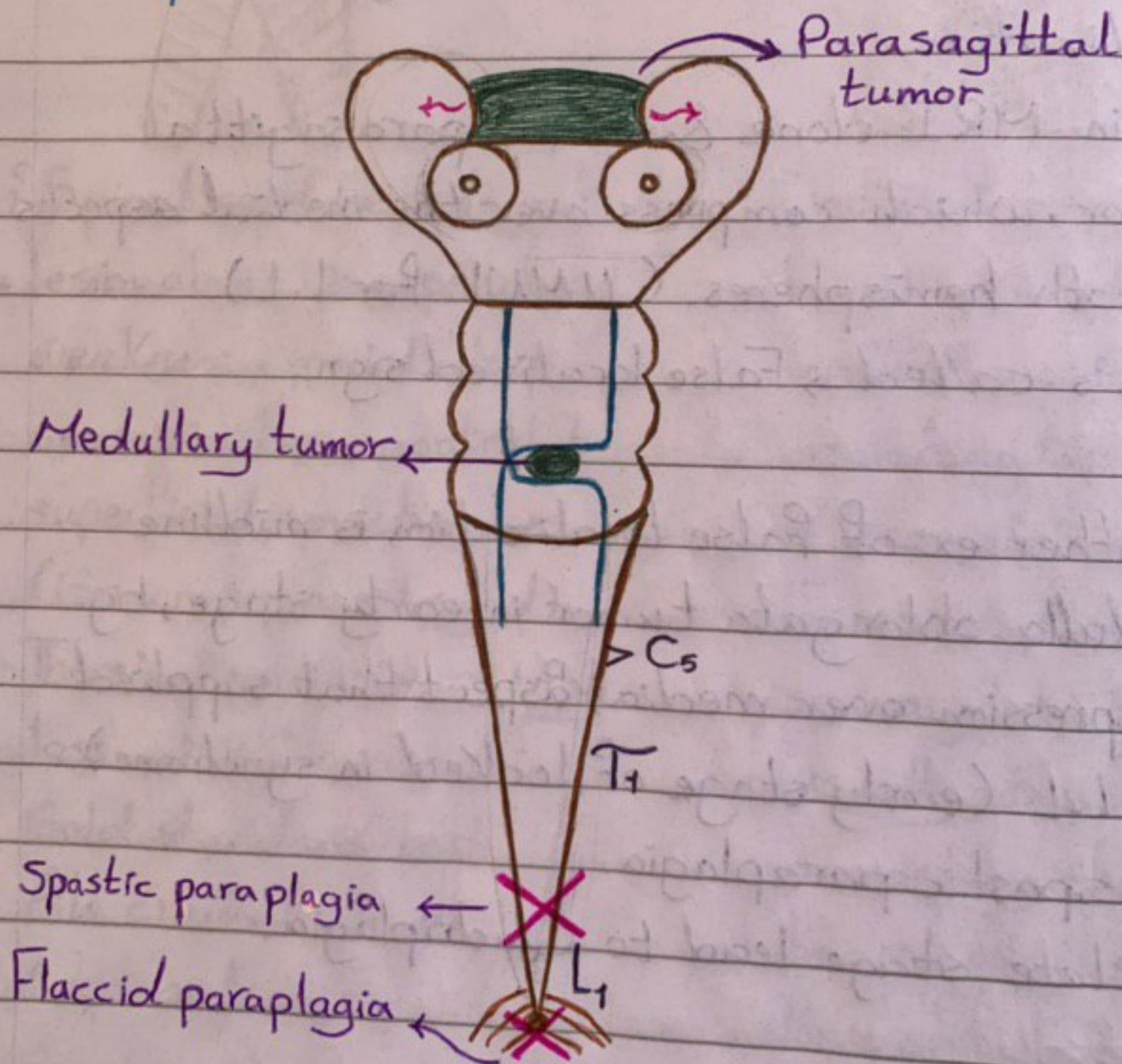
2. Graphesthesia → pt can recognize which number is written in his hand while his eyes are closed.

3. Stereognosis → pt can detect object while his eyes are closed.

4. Mid zone temperature → Feeling temperature by the dorsum of hand.

2. Visceral sensations: All sensations from internal viscera, reaching the CNS via the autonomic nerves. They haven't well central localization, so any visceral pain is vague & dull aching in nature.

3. Special sensations: Cranial nerves.



- Spastic paraplegia → the lesion is b/w $T_1 \rightarrow L_1$.
Lead to UMN in L.L.

- Flaccid paraplegia → the lesion is below L₁.
lead to LMNL in L.L. "cauda equina syndrome"
- **Case :-** pt have paraplegia with hypertonia & hyperreflexia, but spinal cord MRI shows normal spinal cord.

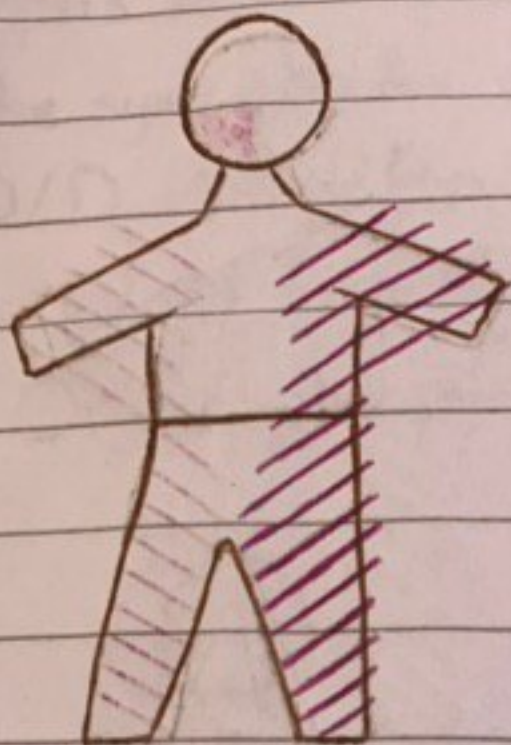
⇒ In this case, examination gives False localization.

Brain MRI done & shows parasagittal tumor, which compress over the medial aspect of both hemispheres. (UMNL For L.L).

This's called → False localized sign.

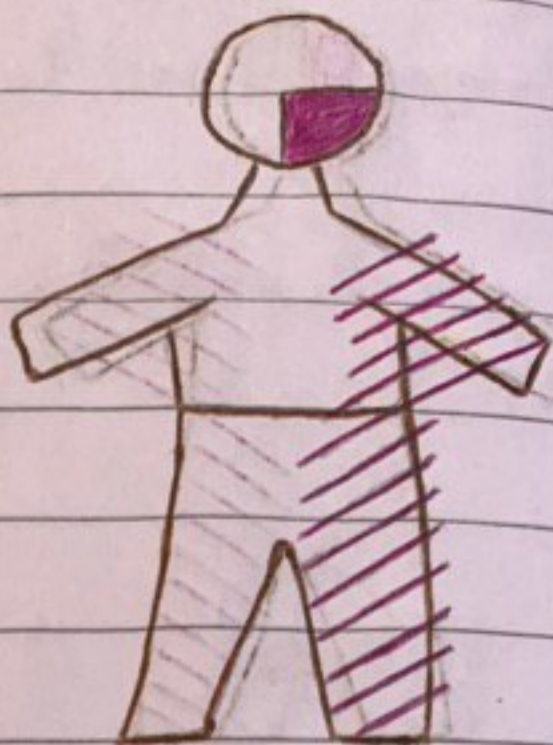
- Another ex of False localization is midline medulla oblongata tumor in early stage, by compression over medial Fibers that supplied the L.L. (early stage of locked in syndrome).
→ spastic paraplegia.

In late stage lead to ~~Quadr~~ Quadriplegia.



{ Spinal cord hemiplegia }

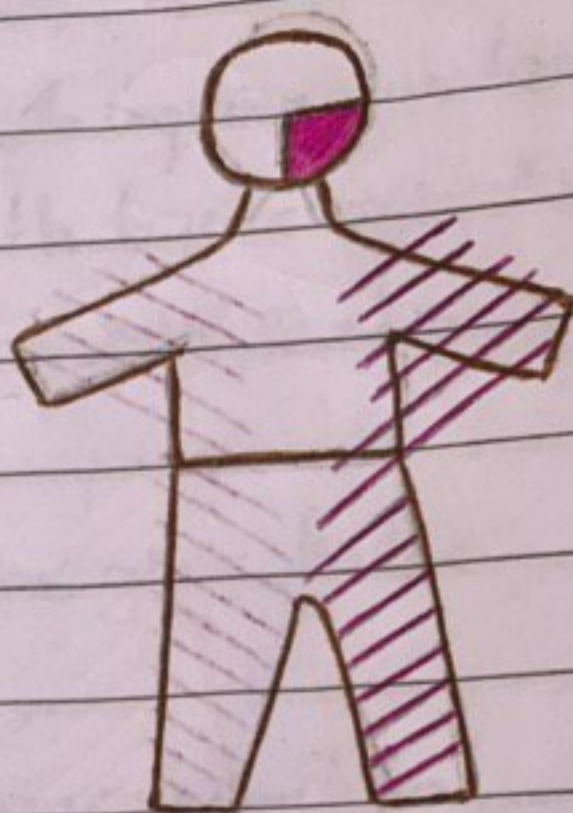
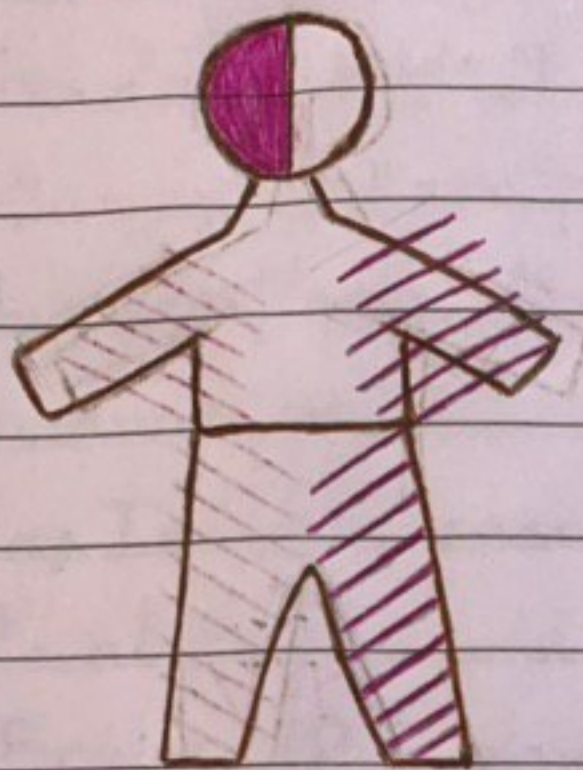
- Lesion above C5 → ipsilateral weakness + ipsilat. deep sensation loss + contralat superficial sensation loss. (Brown squared synd)
- There's dissociation sensory loss → MRI cervical is Gold standard test.
- All cranial Ns are Normal.



{ Cortical hemiplegia }

- have contralateral weakness + cortical sensations loss + loss of localization sense.
- There's no dissociation sensory loss.
- usually starting monoplegia then become hemiplegia. & pt may have convulsion, Aphasia or even coma.
- CN could be normal or affecting lower half of facial nucleus.

We can differentiate b/w Spinal & cortical hemiplegia by Hx & Examination



{ Brain stem Hemiplegia }

(Crossed Hemiplegia)

- Lead to contralat.

hemiplegia + contralat

hemianesthesia (Deep +

superficial) + ipsilateral

CN involved.

(LMNL in CN & UMNL

in upper & lower limbs).

{ Capsular Hemiplegia }

(Non crossed Hemiplegia)

- Lead to contralat.

hemiplegia + contralat

hemianesthesia (Deep +

superficial) + contralat.

CN involved $\rightarrow$ 7, 12

(UMNL) $\rightarrow$ lower half

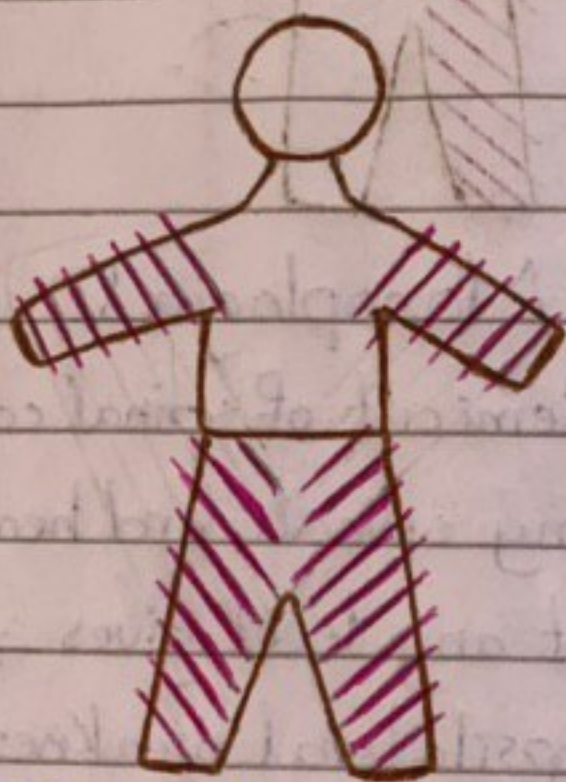
facial weakness.

Note: We can differentiate b/w Midbrain & pons lesion by pupil size, midbrain lesion as by surgical cause of 3N. palsy (Trauma, Tumor, etc) will affect parasympathetic $\rightarrow$ lead to mydriasis.

Pons lesion lead to PPP because affecting the sympathetic chain + part of horner's syndrome.

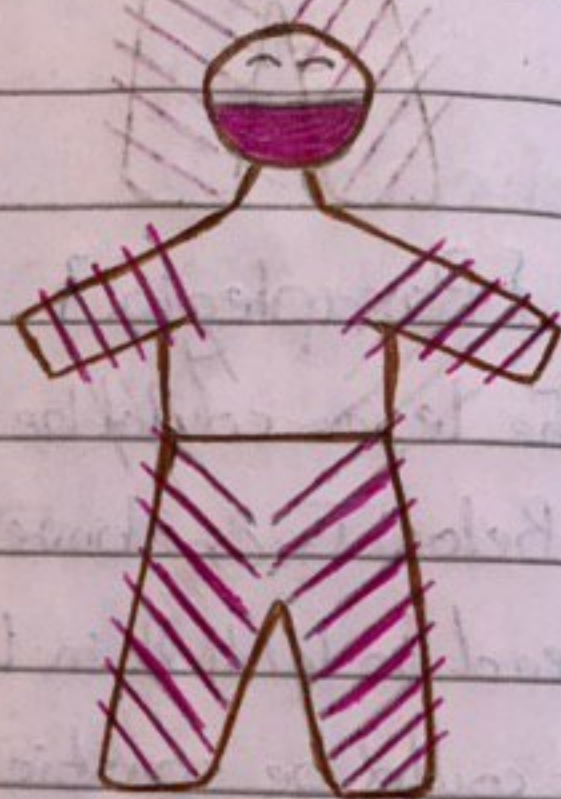
D/D of PPP:-

1. Opioid toxicity.
2. Pontine Hge.
3. Organophosphate poisoning.



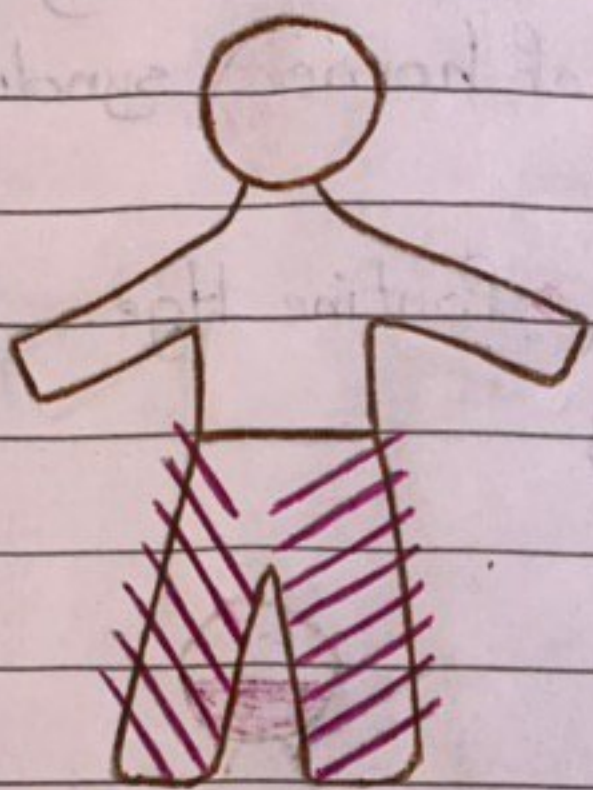
{ Quadriplegia }

- complete cut in spinal cord $\rightarrow$ above C5.
- Below the level of cut all sensations are lost.
- Cranial Ns are normal. (normal face).



{ Locked in syndrome }

- Quadriplegia with bilateral face involvement.
- Brain stem affected bilaterally.
- All cranial nerves involved except oculomotor N.



{ Paraplegia }

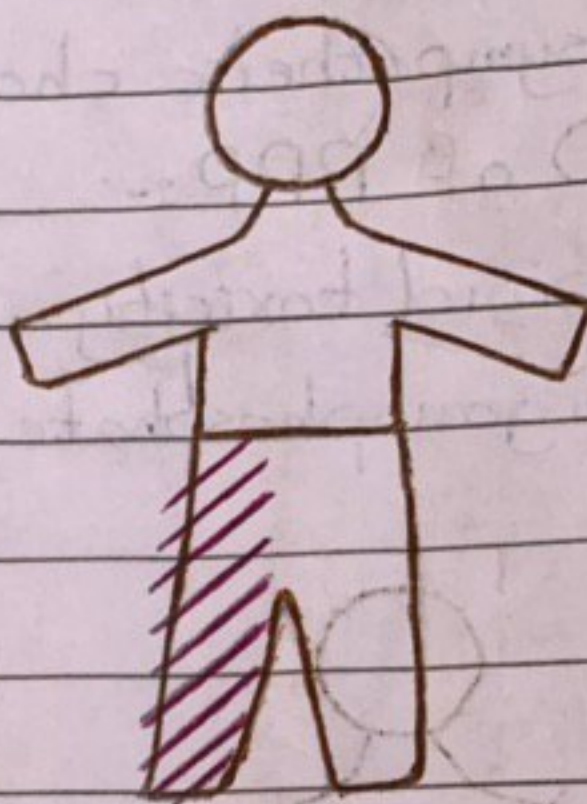
- The lesion could be:

1. Below T₁ & above L₁
lead to UMN in L.L

→ spastic

paraplegia or parasagittal tumor associ. with urine incontinence, headache & projectile vomiting.

2. Below L₁ lead to LMN in L.L → Flaccid paraplegia "quadriparalysis syndrome".



{ Monoplegia }

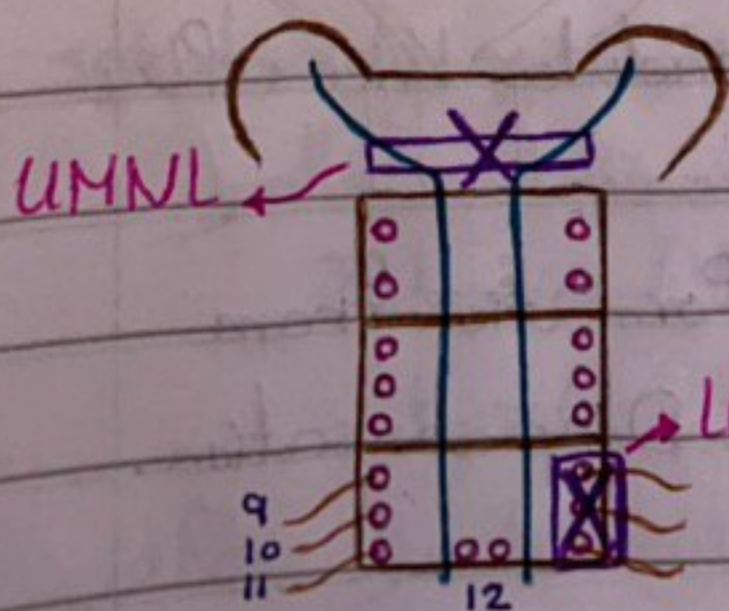
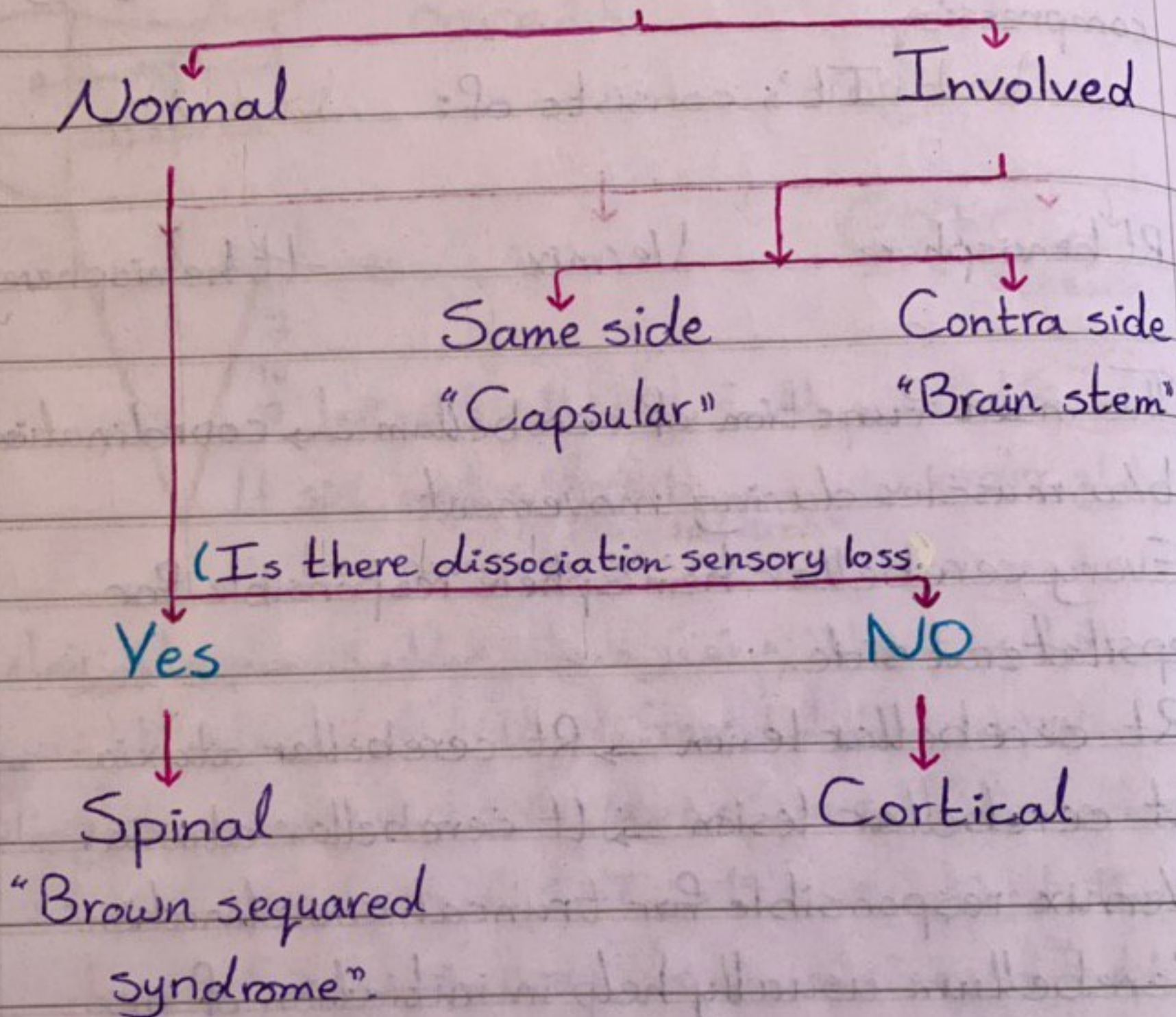
(Hemicut of spinal cord)

- Any spinal cord hemicut at any level gives → ipsilateral weakness, ipsilat. deep sensation loss + contralat. superficial sensation loss (Brown sequard syndrome).

* So this syndrome is termed for any hemicut of spinal cord at any level → either monoplegic or hemiplegic.

Summary of Hemiplegia :-

What's about CN



Note :- Bulb = Brain stem.

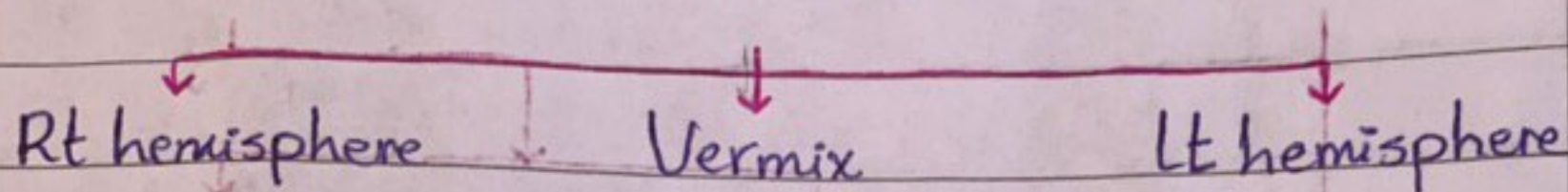
• Any lesion in LMN of CN 9, 10, 11, 12 called bulbar palsy.

• Any lesion in UMN of these nerves called pseudobulbar palsy.

Cerebellum

- It's present in posterior cranial Fossa (posterior to the pons). narrow space, affected easily by compression.

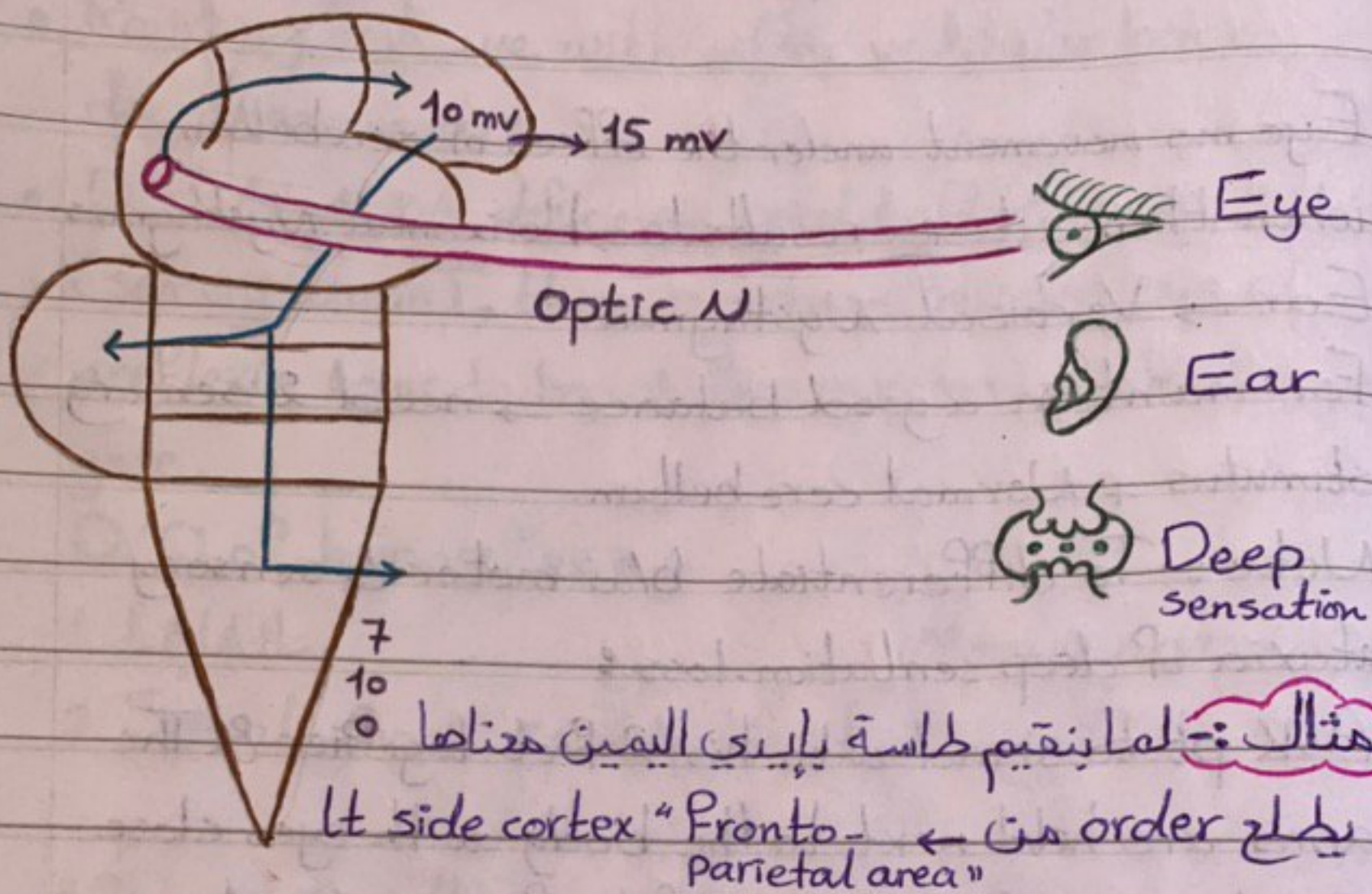
It's consists of:



- The main Function of cerebellum $\rightarrow$ coordination b/w muscles during movement.
- Every cerebellar hemisphere responsible for ipsilateral side.
- Rt cerebellar lesion $\rightarrow$ Rt cerebellar ataxia.
- Lt cerebellar lesion $\rightarrow$ Lt cerebellar ataxia.
- Vermix responsible for truncal coordination.
- Cerebellum usually help in initiation of ms tone, so in some cases cerebellar lesion lead to hypotonia.

& some cases maybe have pendular Knee reflex.

Note: Cerebellum receive information from 3 sensory stimulus: Eye, Ear & Deep sensation.



بس قبل ما نقيّمها لازم نقترّ مسافة الطاسة وحجمها ووزنها،
 فال occiput يقول لل Frontal الحركة الي مقبل عليها الشخص
 بحاجة مثلاً الي 10 mv، ف Frontal lobe يدير حسابه ويطلع بزاي
 15 mv. لو نزلت هذه 15 من CST إلى العضلة حتمشي لبعده
 الطاسة ولو قمتها حنقيّمها over وتسمى Dysmetria.
 لك 5 الزايدة يختزلها Cerebellum فتصبح 10 mv وتنزل
 على النقطة نفسها.

وممكن يجي قبل النقطة لما Cerebellum نفقده، ف spinal cord
 يعوّض ويختزل مرة 7 ومرة 10 ومرة ما يختزل شيء قال مريض
 تكون حركته متزودة وتسمى Intentional tremor
 (Kinetic tremor)

لو خدي أكثر ينزل Prepoint ولو ما خدش بكل ينزل after.

- Eye ms movement under the effect of cerebellum when there's lesion results to → Horizontal Nystagmus
Ear → Vertical Nystagmus.
- For maintain a good balance → need 2 sensory stimulus + Normal cerebellum.

Note :- To differentiate b/w motor & sensory ataxia of deep sensation loss :

- Ask pt to stand with his 2 feet together & The arms are held next to the body with eyes close or stay in dark room → if he feel unsteadiness → have sensory ataxia & this test called → Romberg test. (positive)
- Motor ataxia → pt feel unsteadiness even with open eyes.
- If pt has a defect in hearing, but no problems in eyes & Deep sensation → he can walk with balance, but in resting position will have a vertigo "sense of rotational movement in absence of real movement". especially if moved his head on right or left side.

- Romberg test +ve with either vestibular loss or deep sensation loss.
- DM & vit B₁₂ deficiency Lead to deep sensory loss. (sensory ataxia), these pts have hyporeflexia or areflexia (due to loss of deep receptors) with stumping gait.

D/D of hyporeflexia:

1. LMNL.
2. Spinal shock "sudden onset of LMNL".
3. Sensory ataxia.

Note: When there's weakness "paralysis" we can't examine the cerebellum, because if pt have signs of weakness we can't differentiate it's due to ataxia of cerebellum lesion or weakness (due to ↓ in power).

Ataxia:- Inability of the muscles to coordinate during movement in absence of paralysis.

If your doctor asked you to do motor examination of L.L → Don't do romberg test. *

Coordination examination is different from Cerebellum examination, because coordination examination include: Cerebellum + Basal ganglia + sensory examination for ataxia.

By doing all steps of cerebellum examination + heel to shin test + but here with closed eyes for sensory ataxia + Romberg test.

Types of ataxia :-

1. Motor ataxia → cerebellum lesion (Horizontal Nystagmus).
2. Sensory ataxia → posterior column sensation loss (Deep sensation).
3. Vestibular ataxia → Features of ear problem as tinnitus, vertigo (Vertical Nystagmus).
4. Combined ataxia → mixed lesion (motor + sensory + vestibular) or also called Global ataxia.
ex: Friedreich's ataxia.

Note :- Bidirectional nystagmus = motor ataxia + vestibular ataxia.

Cerebellar ataxia

- Wide based (Drunking gate)
- Pendular reflex or normal
- Ms. power → Normal.

Sensory ataxia

Stamping gate

hyporeflexia or areflexia

Ms. power → Normal

Causes of motor ataxia

↓

Congenital

Friedreich's ataxia.
 Abeta lipoproteinemia.
 Ataxic telangiectasia.
 Spino cerebellar ataxia.
 (idiopathic degeneration).

↓

Acquired

1. Tumor "cerebro.
pontine angle tumor".
2. Trauma.
3. Infection.
4. Inflammation.
5. Vascular "CVA,
post. circulation".

Other acquired causes :-

1. Toxins → phenytoin toxicity, alcohol for long use.
2. Autoimmune disorders → Lung cancer by para neoplastic syndrome (non metastatic manifestation) or celiac disease → but cerebellum manifestation is reversible with gluten free diet.
3. Nutritional deficiency → especially Vit B₁₂ deficiency.
Hypothyroidism → Reversible with thyroxine.

Investigations of cerebellar ataxia :-

CT scan & MRI → especially MRI for posterior cranial fossa & Multiple sclerosis.
 If MRI is normal → Do TET.

IF TFT is normal do Anti-TTG Ab's For celiac
if +ve do upper endoscopy For villous atrophy.

CXR → if pt have Hx of smoking or hemoptysis
(lung cancer).

& look For vit B₁₂ deficiency → by Methyl malonic acid.

Rx → Rx the underlying cause.

Note:- Vascular & traumatic causes of UMNUL
because of sudden onset, they presented early
with weakness, hypotonia & hyporeflexia → Period
of spinal shock (persists For wks - months).

- In this period pt is Bedridden because of hypotonia
→ ↑ Risk of DVT & Bed sore.
- Spinal shock prolongation indicate poor prognosis.
- Causes of death → Early by aspiration pneumonia
if gag reflex is involved.
Late by Bed sore, Depression & suicide.

*** Additional points in speech abnormalities:

- For good repetition need normal Broca's area, normal Wernicke's area & normal Arcuate Fasciculus.
- Broca's area lesion → Motor aphasia.
- Wernicke's area lesion → Sensory aphasia.
- Arcuate Fasciculus lesion → Conductive aphasia.
(can't repeat). ما يقدرش يعاود الكلام
- All these 3 areas are present in lateral surface of Dominant hemisphere. which's supplied by MCA. so aphasia occurs only if MCA occluded (not with ACA or PCA occlusion).

MCA supply in lateral surface speech center & motor area of upper limb.

Q:- pt Lt handed (Dominant areas in Rt side) presented with aphasia, which artery is occluded?

- A. Lt MCA
- C. Lt PCA
- E. Rt ACA

- B. Lt ACA
- D. Basilar A.
- ✓ G. Rt MCA

	comprehensive	Repetition	Fluency
Broca's	✓	X	X
Wernicke's	X	X	✓
Conductive	✓	X	✓

Notes: Motor aphasia called also expressionless aphasia. & confirmed by asking pt to do motor action (pt can follow verbal order by motor order).

- Sensory aphasia called also word salad aphasia.

- Nominal aphasia → pt can't find the appropriate word to name an object.

it's maybe due to damage in wernicke's area in temporal lobe.

- For normal speech, we need also :-

Normal trigeminal (5th CN) → For jaw movement.

Normal hypoglossal (12th CN) → For tongue movement.

Normal glossopharyngeal (9th CN) + Vagus (10 CN) "mainly"

+ Normal vocal cords.

We need also normal lips, palate, normal cerebellum & Basal ganglia.

Any lesion in one of them lead to → Dysarthria, not aphasia.

Abnormal involuntary movement

1. Tremor $\rightarrow$ Types :-

1. Resting = static tremor $\rightarrow$ occurs during rest due to basal ganglia lesion.
2. Intentional = Kinetic tremor $\rightarrow$ occur during movement due to cerebellar lesion.
3. Postural tremor $\rightarrow$ Flapping tremor (Astrexis) & Fine tremor. $\rightarrow$ لازم نسك، الكيف، والروشن

D/D of Fine tremor :-

1. Sympathetic overactivity "ex: anxiety".
2. Endocrine disorders $\rightarrow$ hyperthyroidism, hypoglycemia, Pheochromocytoma.
3. Drug induced $\rightarrow$ Alcohol withdrawal, Benzodiazepines withdrawal, Salbutamol, theophyllin.

D/D of Flapping tremor :-

1. Liver Failure
2. Renal Failure
3. Resp. Failure II
4. phenytoin toxicity

Notes :- Important to ask about constipation in case of tremor, because constipation $\uparrow$ Ammonia & urea $\rightarrow$ $\uparrow$ hepatic encephalopathy.

Central cyanosis + Flapping tremor $\rightarrow$ RF II.

Renal failure $\rightarrow$ $\uparrow$ urea $\rightarrow$ uremic encephalopathy.

2. Chorea: Rapid involuntary movement (Dancing like movement), in proximal $>$ distal due to basal ganglia lesion (Caudate nucleus).

3. Athetosis: Sudden involuntary movement (Dancing like movement), in distal $>$ proximal due to basal ganglia lesion (lenticular nucleus).

Note:- Wilson disease $\rightarrow$ hepatolenticular degeneration, can presented with chorea or athetosis $\rightarrow$ Parkinson like.

Sometimes pt can presented with both 2, 3 (choreoathetotic) $\Rightarrow$ Mixed.

4. Convulsion "Fits": Abnormal movement due to excessive electrical discharge from cortex.

It could be motor, sensory, autonomic, cyclic, visual....

5. Hemiballismus.

6. Tics disorder.

Abnormal gaits

1. Festinating gait (shuffling): Basal ganglia lesion (extra pyramidal) → Parkinson disease.
2. Hemiplegic gait (Pyramidal gait): due to UMN lesion affecting one side of body.
U.L → Flexion & adduction, L.L → extension with circumduction at hip during walking.
3. Scissoring gait (spastic paraplegia): due to UMN lesion in L.L only.
4. Stepping gait: due to common perineal N. lesion (Foot drop).
5. Stumping gait: due to sensory ataxia (Deep sensation loss).
6. Ataxic gait (Drunking or broad based gait): due to motor ataxia (cerebellum lesion).
7. Waddling gait: occurs with any proximal muscle myopathies as Dermatomyositis, Polymyositis, myotonic dystrophica, M.G., acromegally etc.

{ سبجانتك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم }